XLIX No. 2 FEBRUARY 1954

VETERINARY MEDICINE

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features in this issue...

24th Florida State Veterinary Medical Association Meeting

Displacement of the Bovine Abomasum

Mastitis—Etiology, Diagnosis, Control

Newer Treatments in Cattle Practice

Nutritional Deficiency Diseases

Treatment of Ketosis in Dairy Cattle

Value of Nitrofurazone in Pig Enteritis

Small Animal Clinical Laboratory Diagnostic Procedures

New General Anesthetic for Small Animals

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Florida State Veterinary Medical Association

COMPARED with meetings of former years, the 24th annual convention of the Florida State Veterinary Medical Association in Daytona Beach, Oct. 18-20, 1953, maintained — perhaps even exceeded — high standards this group has set for itself. For those fortunate enough to attend, it was a profitable three days.

Dr. E. F. Thomas, President

The enthusiasm for learning among members of this relatively new association exemplifies the real purpose of a state professional organization. Successful meetings of the Florida group, year after year, are not alone due to leadership furnished by selected members, but also to sincere efforts of those charged with responsibilities, and the cooperation of all members of the association. It was noted in the records of this meeting that over 40 Florida veterinarians contributed to the literary program and more participated in discussions and free exchange of practical information.

In this issue of VETERINARY MEDICINE is presented most of the formal program including papers presented by out-of-state speakers. In addition, records of the informal Sunday afternoon session, a feature of Florida meetings, are presented in so far as practical, under the general heading of Short Subjects and Clinical Briefs. Dr. C. E. Bild, Miami, was in charge of this latter session which attracted, as usual, a large, attentive crowd. A total of 42 speakers were scheduled for this session. Each was allowed five minutes by the stop-watch to present his brief and give any favorite procedure or formula found useful in his practice.

President's Address

In his address at the opening session, Dr. Ronald F. Jackson, St. Augustine, referred to failure of efforts to obtain state funds for establishment of the projected state diagnostic laboratory. He recommended, however, that,

Dr. Robert P. Knowles, Secretary

because of the vital importance of this project to improved service to the livestock industry, efforts be continued to encourage consideration of the value of such an establishment. He referred also to practitioner participation in disease-control programs and to progress along these lines in cooperation with state and federal agencies.

Doctor Jackson spoke of the changing economic situation and its influence on veterinary practice. He stated that, "The overhead of the practicing veterinarian in 1953 was vastly greater than that of his predecessor." Greater cost of education, more and improved equipment and supplies, including such items as oxygen dispensing apparatus, *x*-ray machines, blood transfusion equipment, and new and costly pharmaceuticals, biologicals, and antibiotics, add significant costs to veterinary service. Many clients today expect to pay the

same fees that obtained 15 years ago, and some veterinarians continue to make an effort to operate on this basis. The only way that fees can be kept at 1938 levels is by rendering 1938-type services. "If it comes to a choice", Doctor Jackson stated, "I recommend that services be improved in every possible way though it means increasing professional fees. We, as a group, must make our clients and the public generally conscious that they are receiving better service. As a group, we have been prompt in utilizing new drugs as they are made available, and in pioneering new technics, but we have lacked imagination in our public relations efforts. We have been too conservative. We need now a more daring approach to the subject."

The president suggested an enlarged public relations program on a statewide basis in cooperation with the national association. He also recommended serious investigation into health insurance plans for all classes of animals, the withdrawal of state and federal agencies from all but supervisory and regulatory activities in disease control, for recognition of specialization in veterinary practice, for standardization and inspection for all veterinary hospitals as to adequacy of equipment, personnel, and sanitary practices, and a study of possibilities for improved service that might be effected through group practices as briefly outlined recently by Dr. Robert P. Knowles of Miami (VETERINARY MEDICINE, 47:136 [Apr.] 1952).

Those in attendance at all literary sessions complimented speakers, as well as those in charge of program arrangements. Participation by members in these meetings and in the business session indicated interest of membership in veterinary medicine in Florida. The warm fraternalism evident at social functions arranged by the local committee characterized the entire convention, giving emphasis to the stated objective of this association to make veterinary service most valuable to the growing livestock industry of Florida and to the pets of Floridians and their guests.

Election of Officers

Officers selected to represent the Association for the coming year included the unanimous election of Dr. E. F. Thomas, Sarasota, as president, and Dr. Jack O. Knowles, vice-president. Dr. H. L. McGee and Dr. Robert P. Knowles, both of Miami, were reelected treasurer and secretary respectively.

▼ ▼ ▼

The American Cancer Society announced recently that Dr. Ivor Cornman, George Washington University scientist, had isolated a cancer-attacking subtsance from a by-product of early penicillin strains.

Observations made ten years ago indicated that early, unrefined forms of penicillin had lethal effect on cancer cells, yet were apparently harmless to healthy tissue. As production methods for this antibiotic were perfected and refined, the anti-cancer factor was lost.

Clues furnished by earlier work are now being pursued to establish definitely the value of recoverable substances from these old strains of penicillin.

Officers of the Florida State Veterinary Medical Association

(L. to R.) Dr. Robert P. Knowles, Miami, Secretary; Dr. E. F. Thomas, Sarasota, President; Dr. H. L. McGee, Miami, Treasurer; and Dr. Jack O. Knowles, Miami, Vice-President.

Parasites and Parasitic Diseases

Report of Florida State Veterinary Medical Association Committee on Diseases and Research, C. F. Simpson, D.V.M., chairman.

Prevention. (1) Plant improved pastures, graze tall grasses, segregate sick animals and pasture age groups; do not mix ages and/or sexes; divide pastures into small acreages, provide clean drinking water, the flow controlled by automatic floats. (2) It is entirely impractical to rotate pastures sufficiently long to eliminate parasites from cattle, sheep, etc., as it takes from 10 to 12 months to eliminate parasites from an area. However, rotation of short duration is beneficial. Keep animals on adequate balanced diet; provide minerals free choice. (3) Avoid deep shade, low wet areas; provide adequate drainage, cap or control artesian well flows.

Treatment. (1) Administer, either by bolus or drench, phenothiazine according to the dose schedule. In general, the dose of phenothiazine for cattle is 10 gm. per 100 lb. live weight. Do not treat just visibly sick animals, but treat all animals in that particular herd and repeat treatment in 21 days. Furthermore, it is advisable to continue this treatment in the fall and spring each year, especially animals under 18 months of age. (2) The Parasite Laboratory (Gainsville, Florida) has carried out a low-level feeding experiment with phenothiazine N. F. which was completed on December 1, 1953. The report and final results thereof will be compiled and reported in a national veterinary journal at the completion of the project. This report will cover autopsies on approximately 20 head of cattle, checking for any parasites (macroscopic and microscopic) present. It will include an analysis of body tissues for the presence or absence of phenothiazine. The question of adding or feeding phenothiazine to animals is not conclusive, but we will have sufficient evidence at the conclusion of the low-level project to make recommendations. We have found that cattle will not consume sufficient phenothiazine when mixed with salt or minerals alone, in fact these animals have not taken sufficient minerals to satisfy body requirements. In the beginning of the experiment, they had to be force fed minerals for ten days before near-normal consumption of minerals resulted. They will, however, under our limited experiments, take low-level phenothiazine mixed with feed, such as ground concentrate, molasses, cotton seed meal, or a mixture of cotton seed meal and salt. Weights, parasite checks, larvae checks, feed consumed, *et cetera,* will be reported in the final analysis. It is important to state that the control groups

Officers of the Women's Auxiliary

Standing (l. to r.)—Mrs. P. O. Roy, Jacksonville; Mrs. Robert P. Knowles, Miami; Mrs. D. A. Sanders, Gainsville; Mrs. Leroy T. Fisher, Hialeah. Front row (l. to r.) Mrs. George L. Lewallen, St. Petersburg; Mrs. John R. Wells, Palm Beach; Mrs. C. E. Bild, Miami; Mrs. Karl R. Owens, Gainsville; and Mrs. Jack O. Knowles, Miami.

have done as well or better, than the low-level animals, even though egg and larvae counts were higher in the controls. (3) Administer hexachlorethane, as a drench, in doses of 10 gm. per 100 lb. live weight, to all animals in herds where liver fluke infection is found. Repeat in 21 days, as the drug does not destroy the immature flukes. Repeat periodically. In conjunction with hexachlorethane treatment of cattle, drain pastures, control water, and treat all snail infested areas with copper sulfate to destroy the intermediate host snails. (4) Limited experiments have shown that phenothiazine and hexachlorethane can be mixed and given in a single drench. Apparently this mixture is well tolerated in cattle with a proper mineral balance; however, it is detrimental if the animals are mineral deficient. Young calves which are heavily parasitized and weak do not tolerate this mixture, as we have had death losses in calves under three months of age. (5) There are a number of parasites of cattle which are not removed by the two drugs mentioned above; however, the most pathogenic ones are removed, allowing the animals to overcome the other infections. Research is in progress to find drugs and methods to remove all parasites from the animal without injury to the host. Parasites in this group include lungworms, Cooperia, hookworms, tapeworms, and Strongyloides.

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Coccidiosis

A recent report by Georgia public health investigators describes two cases of human infection with *Isospora hominis* which were thought to have been caused by an infected dog in the household. The disease in one of the persons was characterized by numerous stools, by as many as 25 on some days for a 34-day period. A daughter in this household had no symptoms, but fecal examinations revealed parasites. A survey of the animals on this farm demonstrated that the dog was positive for *I. hominis*. Coccidiosis infection in dogs usually is due to *I. felis*.

Coccidiosis is not a rare infection in dogs but it is seldom reported in man. The reason for the low incidence is not known for a certainty but it is probable that human cases frequently are overlooked because the cysts do not appear in feces until the symptoms have disappeared.

The investigators concluded that additional study is needed to determine if the dog is a source of infection for man.

—*J. H. Steele, D.V.M.*

In human diagnostic laboratories, the frog test (*Rana pipiens* test) for pregnancy is looked upon with favor. It is based on the fact that chorionic gonadotropin appears in the urine and serum of pregnant women between the 22nd and 25th cycle day. The urine or unoxalated serum is injected into male frogs; in from one-half to three hours after the injection, if the human being is pregnant, sperm appear in the urinary bladder of the frogs. The test is effective as early as the 30th cycle day, although the 35th day is more certain.

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Recently there has been a good deal of discussion relative to the possible relationship existing in man between the clinical use of chloramphenicol (chloromycetin®) and the appearance of aplastic anemia. It is interesting to note that in a recent research project in swine reporting on the addition of "100 mg. of chloromycetin per kg. of dry matter consumed"—the basal ration of the baby pigs used in this project was the alpha-protein synthetic milk—there is no mention of the appearance of aplastic anemia. Pigs from 48 hours of age to presumably weaning time remained in apparent good health. Since, however, the purpose of the project was in a measure to determine the effect of the antibiotic on the intestinal flora, perhaps no attention was paid to any incomplete or defective formation of tissues as is observed in aplastic anemia.

▾ ▾ ▾

Bivins of Rutgers University, according to an editorial in the June 20 issue of the *Journal of the American Medical Association*, has apparently succeeded in cultivating a virus derived from the common wart. Veterinarians have been handling successfully for many years bovine cutaneous papillomatosis by means of wart vaccine. It was determined several years ago that "bleeding warts" of cattle can be transmitted by inoculation, and practitioners contend that transmission also occurs following direct contact between affected animal and susceptible host. Cultivation of a virus in man adds to our knowledge.

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Displacement Of The Bovine Abomasum†

G. R. MOORE,* D.V.M., M.S., W. F. RILEY,* D.V.M., M.S., R. W. WESTCOTT,* D.V.M., and G. H. CONNER,* D.V.M., M.S., Ph.D., East Lansing, Michigan

DISPLACEMENT of the abomasum is one of several obscure conditions that are of chief importance because they cloud and confuse diagnosis of digestive and metabolic diseases in cattle practice.

In this condition, the abomasum is displaced from its normal position on the abdominal floor posterior to the omasum and in relation to the right ventral aspect of the rumen. Its greater curvature slips under the rumen, comes up along the left side, and is impounded between the rumen and the left abdominal wall.

History

This anatomical displacement of abdominal organs was first observed by us in the fall of 1948 in the course of an exploratory laparotomy. Begg[1] reported two cases and Jones[2] reported three cases in England.

Since our first case in 1948 we have, in the course of performing 582 laparotomies, observed 33 different animals with displacement of the abomasum. The purpose of this report is to record our observations of etiology, symptoms, diagnosis, and surgical treatment.

Etiology

The cause of this displacement is not well understood, but in 31 of 33 cases recorded, the onset of symptoms was associated with the late stages of pregnancy, or began within two weeks following parturition. In the other two, onset followed immediately after an attack of postpartum paresis. In both cases, the cows had been down in an awkward position and had been rolled over on their backs to get them in a more advantageous position for treatment. One can readily imagine that rolling a cow on its back could allow the abomasum to slip under the rumen. Since the postpartum paresis and displaced abomasums in these two animals also occurred shortly after parturition, it is possible also that the relationship to rolling the cows was purely coincidental.

In operating these cases, it was attempted in each instance to make as thorough an exploration of the abdomen as possible in the hope of discovering any cause for the displacement.

Our records in 11 instances reveal the fact that the pregnant uterus had descended under the rumen instead of being deflected to the right, as is considered normal. We believe that this phenomenon was a factor in at least part of our cases. A uterus in this position, in addition to elevating the rumen, also would tend to push the abomasum forward and cause it to double up. If true, this should at least develop a tendency for the greater curvature of the abomasum to slip under the rumen. One also can readily visualize that this same set of circumstances could occur during the act of parturition. The fact that all of our cases occurred in cows that had given birth to one or

†Presented at the Florida State Veterinary Medical Association annual meeting, Daytona Beach, Oct. 18-20, 1953.

*Large Animal Clinic, Department of Surgery and Medicine, Michigan State College.

more calves and that the symptoms developed during the last two months of pregnancy, or soon after parturition, strongly supports this hypothesis.

Anatomy

Anteriorly, the abomasum is attached to the rumen, reticulum, and omasum by folds of peritoneum. It lies on the abdominal floor posterior to the omasum and to the right of the rumen. Its posterior portion, near the pyloris, is suspended from the liver by the lesser omentum. The central part, particularly along the greater curvature, is free to undergo considerable variation in position.

In all of the cases of abomasal displacement observed by us, it was quite obvious that the organ had doubled upon itself so that the lesser curvature was abrupt and the greater curvature was greatly exaggerated. It also was quite apparent that this latter was the portion that had slipped under the rumen and then dorsally between the left rumen and abdominal walls.

Another interesting feature observed in all of our cases was that the part of the abomasum thus impounded had become greatly enlarged by distention with gas and liquid ingesta. In a few cases, it was nearly as large as the rumen; in all it appeared to be at least double normal size. There appeared to be quite a close correlation between the degree of enlargement and the duration of symptoms.

Symptoms

In looking back over our records of displacements of the abomasum, it is now apparent that they followed quite a definite pattern. We feel that we can now diagnose these cases with reasonable accuracy.

Initial symptom observed was sudden diminution or cessation of appetite. After a few days appetite was intermittent but always poor. In all cases, the cows showed a preference for hay with no appetite at all for grain or ensilage. As a rule they ate only enough to keep alive.

Perhaps the most constant symptom observed was a mildly positive urine ketone test. On the basis of 4+ for a good strong reaction, these cases were all rated 1+ or 2+ by our diagnostic laboratory. At least half were referred by practitioners who had previously treated them for chronic acetonemia without success.

Other constant symptoms observed were scanty feces of a soft, pasty nature, a dull listless attitude, and a tucked-up appearance which was not always obvious prior to parturition. Milk flow was greatly diminished and loss of weight rapid.

In all cases except one, the temperature remained normal or slightly subnormal. This one case, in which a temperature of over 103 F. was noted on three successive days, was found, when operated, to be suffering from an accompanying traumatic reticulitis. This patient also had a total white cell count of 18,600 with a marked increase in the percentage of neutrophils, and a shift to the left of Schilling's index. In the other 32 cases, both total and differential blood cell counts were within the normal range.

As far as we are able to determine, displacement of the abomasum seldom, if ever, results in death. Affected animals linger on indefinitely at a subsistence level, but become worthless because of debility and emaciation. One case in particular exhibited symptoms for over a year before surgical correction. This animal was one of Michigan's top Jerseys. It was one of two cows, previously mentioned, that had been rolled during treatment for milk fever. During the ensuing lactation and pregnancy the above described symptoms were present constantly. After calving (one year after onset) this cow was sent to our clinic for examination and subsequent autopsy. A laparotomy was performed and reduction of the displacement effected. Almost immediately appetite improved and the animal made a spectacular recovery. Recent reports from its owners indicate that this cow is well on the way toward a 1,100-pound fat record for the current lactation period.

Diagnosis

Diagnosis is based on history and symptoms, plus careful differentiation from several other anomalies that present a similar clinical picture such as: (1) Acetonemia—differential diagnosis is based on mild ketone reaction and failure to respond to accepted acetonemia therapy; (2) pyelonephritis—urine studies plus rectal palpation of kidneys and ureters; (3) traumatic reticulitis—absence of temperature elevations, lack of pain on palpation, and normal blood picture assist in eliminating foreign body perforation; and (4) leukosis—absence of leukemic blood picture, enlarged lymph nodes, etc.

Prognosis

In untreated cases, prognosis is unfavorable. Because of difficulties encountered and the force required to reduce these displacements

surgically, we are certain that spontaneous reduction never occurs.

Prognosis is reasonably favorable when proper surgical treatment is used. Of our series of 33 cases, 21 recovered following surgery, including two that were operated a second time because we had either failed to effect complete reduction, or the displacement had recurred. Of the remaining 12, five died and seven were sent to slaughter. In interpreting this rather unimpressive record, one should remember that we knew nothing about this condition when confronted with first cases. Ten of the most recent 11 cases resulted in complete recovery.

Surgical Treatment

Before attempting surgical intervention, one needs to survey the situation carefully. If the cow is near calving and if, in the opinion of the clinician, general condition is such that the patient will survive the waiting period, it is probably best to delay surgery until after parturition. It is next to impossible to effect reduction of the displacement during late pregnancy without first doing a cesarean section.

The surgical approach is through the left paralumbar fossa. For anesthesia we use a standard paravertebral lumbar block. A seven-inch laparotomy incision is made two inches posterior and parallel to the last rib. A quick inspection at this point will reveal the abomasum if the diagnosis is correct. It will appear, as a rule, as a tubular structure six to eight inches or more in diameter curving upward and forward just anterior to the last rib. Its dorsal aspect is usually almost on a level with the top of the rumen. In some cases of long standing, dilatation may be so great that the abomasum fills the whole left flank so that the rumen can not be seen. In this event, it can be recognized by its paler color and thin wall. It also can be pushed forward, exposing the rumen.

In most cases the dorsal part, which is the greater curvature, is filled with gas. Considerable reduction in size can be effected if the gas is allowed to escape by tapping the organ with a 12-gauge needle equipped with a length of rubber tubing to carry gas out through the incision.

If the rumen is loaded with heavy ingesta, as is usual, it is opened and about two-thirds of the contents removed. At this time the reticulum should be explored and any foreign bodies removed. The omasum then should be palpated through the right rumen wall. In this condition, quite frequently, it is enlarged and firmly impacted. If so, it should be kneaded vigorously with the fingers through the rumen wall. Whether or not it is impacted also will have a bearing on the aftercare required. When the above steps have been completed, close the rumen incision with a double row of right angle Cushing sutures.

Pushing the abomasum back under the rumen to its normal position would seem to be easy. It is anything but that. After trying many different approaches to the problem, we have developed a method that works quite well. Two umbilical tape sutures, 18 inches or more in length, are anchored to the left wall of the rumen as far below the incision as possible and the ends are brought out to the exterior. If a steady pull is exerted on the tape sutures, the rumen can be rotated clockwise from its normal position through about a 45 degree arc. Before starting rotation, operator first pushes the abomasum down as far under the rumen as possible. He then holds it there with his hand and forearm while an assistant pulls the tapes to rotate the rumen clockwise as far as possible. The tapes then are released and, as the rumen rotates back to its normal position, it sweeps the abomasum along with it. Usually several attempts are necessary before complete reduction is effected. Three million units of penicillin are then placed directly into the abdominal cavity and the laparotomy incision is closed with a single row of umbilical tape figure-eight sutures, which are removed on the eighth or ninth postoperative day.

Aftercare consists of administration of antibiotics as indicated. Dextrose is given daily in the vein until appetite returns. If the omasum is impacted, mineral oil or rumide® is given on the second day.

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1. Begg, H., *Vet. Rec.*, 63:797, 1950.
2. Jones, E. Eynn, *Cornell Vet.*, 42:53, 1952.

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Three new buildings and adjoining laboratories comprise the new Veterinary Science Research Center at the Ohio Agricultural Experiment Station at Wooster. One of these units is intended for swine and sheep only, the second is reserved for cattle, and the third is an isolation unit for any type animal when disease contagion is a factor.

®Pitman-Moore Laboratories, Indianapolis, Ind.

Mastitis: Its Etiology, Diagnosis, And Control†

G. T. EDDS,* D.V.M., Ph.D., and J. C. TRACE,* D.V.M.,
Fort Dodge, Iowa

RECOGNIZING that certain significant contributions have been made to our knowledge of mastitis, continuing economic losses make reports of new discoveries of significant concern to the dairy cattle industry. Continuing interest in this important disease throughout the world may be seen readily by examining many current professional veterinary journals. Publication of newer methods of diagnosing mastitis, as well as recent reports on pathological changes which are associated with clinical mastitis, have enabled veterinarians to interpret better the results obtained from treatment. A review of some significant factors influencing diagnosis and treatment of this infectious disease may be of interest.

Bovine mastitis is defined as an infectious disease of the udder, characterized by inflammatory changes and accompanied by physical and chemical alteration of the properties of secretions. The immediate problem of the attending veterinarian is to properly diagnose, treat, and return the dairy cow to normalcy and full milk production. Both acute local mastitis with limitation of infection in the udder, and acute systemic mastitis, accompanied by systemic symptomatology resulting from toxemia and bacteremia, are of vital importance.

Etiology

Introduction of pathogenic microorganisms after such predisposing factors as teat and udder injuries resulting from poor milking technic or improper stabling, generally lead to production of clinical mastitis. The veterinarian also should be sufficiently aware of the several types of milking machines on the market and the advantages and disadvantages of each, to enable him to discuss their proper uses with dairymen clients. Faulty milking machines or milking technics have been credited with causing more mastitis than any other single factor[1].

Baker and Bailey[2] recently proposed that every up-to-date manager should follow a rapid milking procedure, thus reducing machine time and total milking time. This includes massaging the udder with warm water and drawing one stream of milk from each teat into a strip cup one minute before application of the milking machine. The machine should be removed as soon as the cow is milked out, usually in four minutes or less. These suggestions have been proved physiologically sound, because: (1) Cows must be milked out completely or drying will set in; (2) the letdown of milk is associated with the massaging of the udder and release of pitocin which causes relaxation of the smooth muscle surrounding the ductules allowing, in turn, alveoli to empty into the milk cistern; (3) it has been proved that excitement interferes with milk letdown by reason of epinephrine being liberated into the blood stream with relaxation of the smooth muscle surrounding the alveoli; (4) once letdown has been stimulated, cows should be milked out in the four-minute period or a portion of the milk secreted is retained; (5) cows milked properly should enjoy being milked and even difficult milkers may be retrained to produce larger quantities for longer periods than in previous lactations; and (6) probably most important, leaving the milking machine on the cow's udder is likely to cause injury to the teat lining which predisposes to mastitis. These authors also have suggested additional ways for improving milking conditions and increasing economic returns to dairy farmers.

Other factors influencing the incidence of mastitis include the stage of lactation. There is greater incidence among cows toward the end of the lactation period. As the age of the cow increases incidence is likely to increase, possibly because of infection of one quarter spreading from that quarter to an adjacent one. As mentioned previously, traumatism is probably the most important single predisposing factor.

Diagnosis

A number of methods for detection of mastitis have been proposed, such as the strip cup, pH determination, leukocyte count, catalase test, Hotis test, bacteriological diagnosis,

†Presented at the Florida State Veterinary Medical Association annual meeting, Daytona Beach, Fla., Oct., 18-20, 1953.

*Ft. Dodge Laboratories.

and more recently a modification of the latter by use of containers with selective media for detection of streptococcal and staphylococcal infections.

The use of selective media has proved useful in simplifying cultural procedures. Chapman[3] confirmed the inhibition of organisms other than pathogenic staphylococci by sodium chloride. He noted that coagulase positive staphylococci grew luxuriantly and produced yellow zones on bactophenol-red mannitol salt agar. McCulloch[4] suggested that this medium appeared to be selective for pathogenic staphylococci isolated from cases of chronic bovine mastitis.

Wilson and Slavine[5] described a modified Edward's medium which is placed in wide-mouthed jars. Milk is ejected directly from the cow's teat onto the medium. Lids contain a filter pad insert which is saturated with the sample and from which transfers may be made for further diagnostic tests. In a series of tests on 557 animals, these workers found the jar method to be more accurate than plating on blood agar and plating incubated samples on Edward's medium.

More recently, a jar has been developed which contains one section filled with the modified Edward's medium and one section filled with the salt mannitol agar for bacteriological diagnosis of mastitis. Inclusion of a filter paper insert in the cap of the jar enables one to check also for the presence of coliform bacteria which would not grow on the selective media in the original jar. The sample of milk is collected in jars, the excess is emptied into a waste container and the jar lid replaced. The capped jar is inverted and incubated over night. In those cases of clinical mastitis caused by organisms other than streptococci or staphylococci, negative results after 24 hours incubation suggest culturing of the milk residues from the cap of the bottle for coliform or yeast organisms. There has been a high correlation between the results obtained with these new diagnostic bottles and the presence or absence of clinical mastitis in herds tested. A diagnostic bottle is illustrated in fig. 1.

Pathology

Taylor and Pattison[6] presented an excellent series of articles describing the pathogenesis of mastitis caused by streptococcal infection. In the incipient stages of mastitis, streptococci were found to have penetrated through the alveolar epithelium into the interacinar spaces. These are observed in tissues for 24 to 48 hours, after which they disappear and then are found only within the ductules and alveoli. Streptococcal mastitis generally affects isolated lobules, those areas recover and the infection spreads by way of the duct system to adjacent lobules. The involvement of one lobule with no infection of the adjacent one is illustrated in fig. 2. These progressive lobular infections explain the chronic characteristics of streptococcal mastitis and account for the extensive fibrosis following repeated flare-ups.

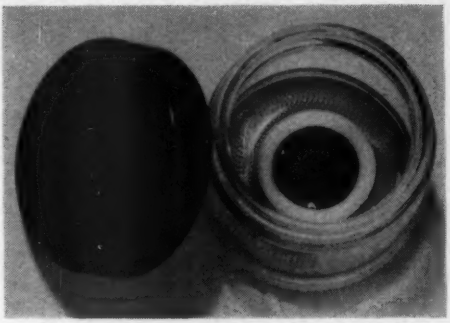


Fig. 1. Central area plated with Edward's medium. Peripheral area plated with phenol-red mannitol salt agar.

Sections of bovine udder tissue from cows with chronic mastitis were examined for the presence of microorganisms, but none were observed. Previous workers,[7] however, have reported the presence of staphylococci in the interacinar spaces in cases of chronic mastitis. Since antibiotic level after treatment with such agent is appreciably lower in this interacinar tissue space than within the alveoli, and since staphylococci are present in this area, poorer therapeutic results are obtained than in those cases in which the organisms are found only within the acini. An important alveolar change accompanies these repeated attacks, that is, involution of the glandular area. The secretory tissue ceases its activity, regresses, and proliferation of the connective tissue fills in where alveoli disappear. (See fig. 3.)

On careful examination of ductules in infected mammary tissue, one finds varying stages of papilliform projections of the lining epithelium into the lumen of the ducts. This has been reported in goats', bitches', and human breast tissue and has also been observed in our own laboratories in bovine tissue. These papilliform projections may completely occlude ductules during certain of the early phases of mastitis and thus block the passage

of therapeutic agents into particular infected lobules. Projections have been observed to regress within a few days and this again would allow the passage of therapeutic agents for successful treatment. Therefore, rational therapeutics should be followed by improved results if the level of the drug within the udder is maintained for five to six days by repeating the administration of therapeutic agents at the proper time.

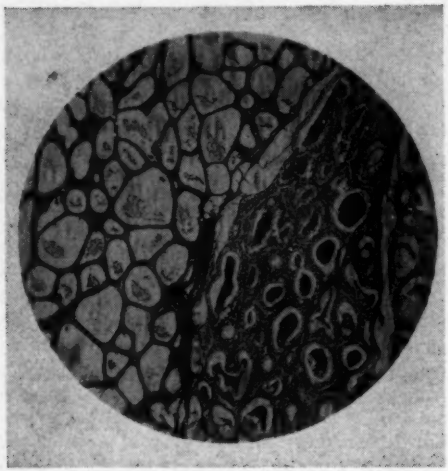


Fig. 2. Microscopic view of mastitis infection illustrating involvement of one lobule, with no apparent infection of adjacent lobule.

Treatment

In the development of new drugs, veterinarians, livestock owners, and manufacturers are concerned that the drugs be proved safe, non-toxic, show no irritation or cause other tissue damage, and in addition, be proved effective both in the laboratory and in clinical practice. In a report released by the Committee of Animal Health, National Research Council,[8] the value and limitations of intramammary therapy were discussed with summaries of results obtained in controlled investigations on each of the chemotherapeutic and antibiotic agents available for treating bovine mastitis. Most investigators were in agreement that mastitis due to *Streptococcus agalactiae* was curable by use of a number of sulfonamides and antibiotics now available.

Use of penicillin alone in treating bovine mastitis was reviewed by Jepson[9]. He suggested that penicillin is active only against dividing bacteria and, therefore, effective penicillin levels should be maintained as long as these bacteria divide. This was estimated to be five to six days. Such an effective level could be maintained by giving two doses of 100,000 units of procaine penicillin per quarter at 72-hour intervals. Maintenance of this effective level for five to six days also permits passage of the antibiotic into affected lobules which may have been blocked off temporarily by the papilliform projection into the ductule. As streptococcal mastitis has been more effectively controlled, the incidence of staphylococcal and coliform mastitis has increased.

Certain of the more resistant strains of staphylococci, isolated from clinical cases of mastitis, have been shown to be more susceptible *in vitro* to a combination of penicillin and streptomycin, as well as a combination of these antibiotics and the sulfonamide, sulfisoxazole, than to the single drugs[10]. This synergistic action on these strains of staphylococci is summarized in tables 1 and 2. These favorable combinations of antibiotics have been of value in treating clinical cases of mastitis since some of the resistant strains or organisms isolated from clinical cases now become susceptible to the antibiotic combination. The formula found most effective consisted of:

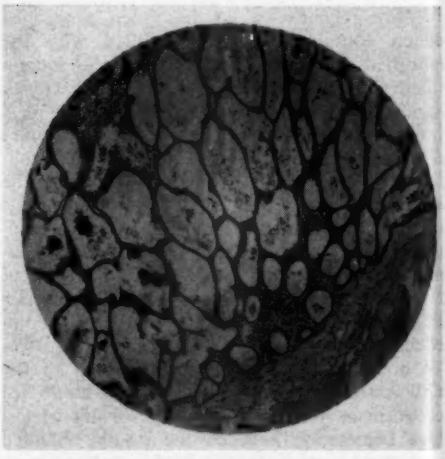


Fig. 3. The replacement of normal secretory tissue is noted in the lower right section in this microscopic view of udder tissue.

Procaine penicillin	100,000 units.
Dihydrostreptomycin	100 mg.
Sulfisoxazole	750 mg.
Sulfathiazole	750 mg.
Peanut oil, aluminum monostearate base, q.s.	10 cc.

In some clinical reports[11] on this formula, clinicians reported that larger doses repeated daily have been effective in clearing previously resistant cases of mastitis due to Pseudomonas organisms. This has included a daily dose of 50 cc. into each quarter.

Pratt and Dufrenoy[12] reported that trace amounts of cobalt enhance the antibiotic effectiveness of penicillin. They demonstrated that traces of cobalt salts added to cultures of *Eberthella typhosa, Staphylococcus aureus* and *Escherichia coli* markedly lower the minimum effective inhibitory concentration of penicillin. They reported also that this property of cobalt is quite specific as salts of all other heavy metals fail to produce this effect.

Similarly, Strait *et al.*[13] found that traces of cobalt produced a two- to four-fold increase in effectiveness of penicillin against *Staph. aureus, E. coli, Proteus vulgaris* and *Bacillus subtilis.* This action was attributed to the marked increase in the rate of bacterial growth in the initial stages of development when the rate of growth is usually slower; that is, the lag phase. Since it is known that penicillin is most effective as a bactericidal agent when the bacteria are in a state of rapid development, the authors explained the improved penicillin action along this line.

TABLE 1. Levels of Antibiotics alone and in Combination Necessary to Inhibit Staphylococcal Strains

Cultures	P	S	P+S*
H263-2	5.0	5.0	0.02
682-4	0.2	0.5	0.05
13-4	0.5	5.0	0.05
12-4	0.5	5.0	0.02
H85-2	0.2	0.1	0.01
E570-4	0.2	0.2	0.02
847-4	0.1	0.1	0.02
H129-1	0.2	0.5	0.02
831-4	0.2	0.5	0.01
643-4	0.2	1.0	0.02

P—Penicillin, concentration of penicillin in units per milliliter causing inhibition of visible culture growth.
S—Streptomycin, concentration of streptomycin in micrograms per milliliter causing inhibition of visible culture growth.
*Combinations showing increased antibacterial activity.

In vivo tests on mice likewise demonstrated that addition of cobalt to penicillin caused a two- to four-fold enhancement of its action. It was found that some of the bacteria, found to be more sensitive to penicillin in the presence of cobalt, belonged to the Gram negative group known to be highly resistant to penicillin. The inclusion of cobalt or the injection of vitamin B_{12} provides quantitative and qualitative promotion of the antibiotic value of penicillin, that is, an extension of the antibiotic spectrum. The value of inclusion of this heavy metal is the attainment of more reliable results where penicillin may have been ineffective in the past.

Parenthetically, Dowling and Leper[14] recommended recently that the clinical effectiveness of penicillin could be potentiated by the inclusion of vitamin B_{12} in those patients with infections resistant to dibenzylpenicillin alone.

TABLE 2. Influence of Sulfisoxazole on the Inhibitory Activity of Penicillin

Strain	Concentration in units or micrograms preventing visible growth: P (units)	SS	P — SS* (units)
H263-2	5.0	+	0.02
682-4	0.2	+	0.02
13-4	0.5	+	0.02
12-4	0.5	+	0.02
H85-2	0.2	+	0.01
E570-4	0.2	+	0.005
847-4	0.1	+	0.02
H129-1	0.2	+	0.02
831-4	0.2	+	0.02
643-4	0.2	+	0.02

P—Penicillin; SS—sulfisoxazole (+) growth occurred in all strains at a level of 50 mg. per milliliter.
*Potentiation of antibiotic activity.

Thus, in 1,303 cases, toxic side effects of penicillin from hypersensitivity were present in 1.2% of the cases. The occurrence was, however, much more frequent with large doses, for here the incidence was 7.8%. Thus, the veterinarian might well combine simultaneous therapy of cobalt or vitamin B_{12} administration along with penicillin therapy.

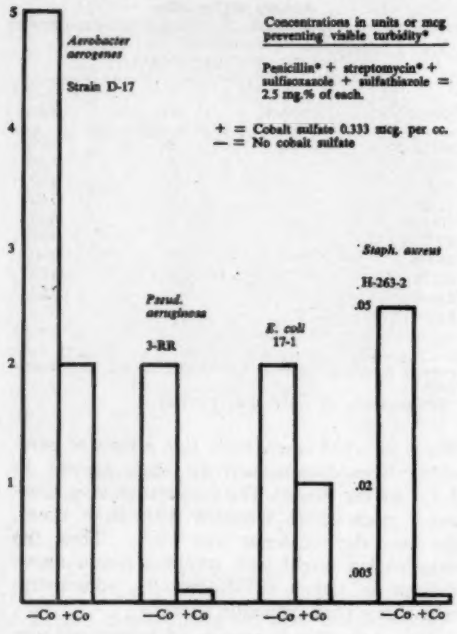
Frequent clinical reports of hypersensitivity of animals to penicillin would indicate that such combined therapy, using cobalt or vitamin B_{12} to potentiate penicillin, would also be important in veterinary medicine.

Prior to the addition of a new drug to a formula which had been giving good results, it was essential to establish the *in vitro* effectiveness of the new combination as well as its safety and toxicity. Strains of the following species of organisms were selected for comparing the level of antibiotics necessary to inhibit visible growth in tryptose-phosphate broth both with, and without, the addition of cobalt. The data included in chart 1 demonstrate the two- to ten-fold increase in bacteriostatic activity when cobalt was added to this antibiotic combination.

Arrangements were made with a local dairyman to investigate whether there would be ir-

ritation and toxicity when cobalt was added to the antibiotic formula being studied when it was infused into the udder of dairy cattle. This herd was selected because the herdsman was known to exercise care in his milking program. The daily milk yields were recorded, and there was no mastitis in the herd. Data collected confirmed the suggestions made by others that addition of cobalt in these trace

Chart 1. Influence of Cobalt on the Inhibitory Activity of a Combination of Antibiotics and Sulfonamides



amounts, although sufficient for enhancement of antibacterial activity of the antibiotics, did not cause any observable changes in the milk production of dairy cattle. Because of improved antibiotic activity, the new formula and the other necessary data were submitted to the Antibiotic Division of the Food and Drug Administration for approval. It is felt that the new combination of antibiotics with cobalt will provide the means for better service to the dairy cattle owner in treating mastitis.

Certain other factors important to better results in treating mastitis might be emphasized in these concluding remarks. Veterinarians realize and should emphasize that poor results may accompany treatment when the lobule is swollen or the ductules are filled with debris and drugs can not penetrate to the site of infection. Note should be taken also of the work of Pattison *et al.* in which they describe the papilliform proliferation of the epithelial linings of the ductules with partial or complete occlusion of the ductules. This knowledge, if imparted to the dairy farmers, would be taken as the basis for repeated treatment in 72 hours to assure the highest possible recovery rate. Furthermore, the work of researchers in the field of dairy bacteriology demonstrating that these bacteria continue dividing for three to five days, makes it necessary that an adequate antibiotic level be continued for that period for maximum effective action.

Tubular debris may be partially or largely removed by using a new technic described by Schipper and Petersen[15]. An aqueous solution of oxytocin is injected intravenously. These workers demonstrated that as little as three units were sufficient; however, they generally propose 20 to 40 units as the proper dose. The smooth muscles surrounding the alveoli and ductules contract, forcing debris along the ductules into the milk cistern. This material is milked out into a waste receptacle and the treatment for mastitis is immediately infused into the emptied quarter. Having eliminated the material blocking the ductules, medicaments spread readily to all sections of the quarter.

The addition of cobalt to the antibiotic combination, with a two- to ten-fold increase in bactericidal and bacteriostatic activity, plus a broadening of the antibacterial spectrum will provide a new and better method of treatment. Proper diagnosis and suggested ways to overcome mechanical obstacles to successful treatment of mastitis, should make possible improved service of greater value.

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(*Continued on page 60*)

Newer Treatments Employed In Cattle Practice†

ADRIAN M. MILLS,* D.V.M., Athens, Georgia

I WOULD like to present a few of the newer treatments we have used, and are now using, at the Georgia Clinic.

A drug that has been in use for several years but that has been shoved into the background by much advertised and more spectacular agents, is neoprontosil.®[1] It is an excellent vehicle for carrying antibiotics. It furnishes a sulfonamide in 5% solution, and, being a thin liquid, is easily injected. For the treatment of mastitis, use about 30 to 40 cc. per quarter to which has been added 100,000 units of penicillin (aqueous) and 0.5 gm. of streptomycin. It is one of the best treatments for streptococcic mastitis. Care must be taken when mixing to avoid staining hands or clothing or both.

Mastitis

Mastitis is one of our worst headaches. Many cases presented have been farmer-treated or mistreated and often are so advanced that nothing can be absorbed through the teat canals of badly swollen udders or quarters. Gangrene is frequently imminent or already evident. Ice packs, intravenous sulfonamides, and systemic antibiotics are indicated if treatment is decided upon. Much nursing care will be required and recovery is slow.

Radical surgery by amputation of the affected teat or quarter, or providing for adequate drainage of the gangrenous area, is necessary and must be performed promptly. The time required to slough out an infected quarter is materially shortened by daily injections of tryptar.®[2] It is a lyophilized crystalline trypsin that digests and liquefies dead tissue. Inject it into the affected quarter in several places, using 200 mg. (one bottle) daily.

Many other agents have been used for relief of mastitis, preference given to those available in tubes. There is no mixing, materials are ready to infuse, and contaminated tubes or syringes are no problem. They have the disadvantage that every farmer can use them; but under present conditions almost anyone can buy any of the antibiotics. I like to use two or three tubes per quarter and massage well. A badly swollen quarter needs more than one small tube of material. If the infusion is made after the night milking, and left overnight, the quarter can be stripped out several times the next day and reinfused the following evening.

Often a cow with acute mastitis and a sensitive udder will not let milk down. One way to clean all milk from the udder thoroughly is to inject 3 to 5 cc. of posterior pituitrin into the jugular or milk vein, wait one minute, then milk the cow. Milk letdown almost invariably follows such injection. This operation can be repeated as often as necessary. Doctor Schipper of the University of Minnesota reported excellent results with purified oxytocin or pitocin. (*Vet. Med.*, 48:401 [Oct.], 1953). An udder engorged with infected milk should not be infused with any treatment until it is empty. Milk dilutes antibiotics and interferes with absorption. It also hinders dispersion through the quarter. Toxic or infected material should be thoroughly removed. Pituitrin will effect the removal unless the udder is acutely swollen.

Retained Placenta

Retained placenta is a serious disease and a difficult one to treat. If a cow continues to eat, and exhibits no symptoms of toxemia, it may be advisable to allow the placenta to slough away rather than attempt removal. This may not please the owner and it invites screw worms. Removal of adherent placental membranes by force, with resulting damage to the uterus and probably to the breeding life of the cow, is not good treatment. Williams stated: "One of the elements of confusion is the belief of cattle breeders that a skillful veterinarian can successfully remove a retained afterbirth at will." There is no doubt that the putrefying mass is better out, and the earlier the better, providing removal does not cause more harm than good.

If removal is attempted, it is best to delay for 48 to 72 hours to allow sloughing. One might give 30 to 35 cc. of stilbestrol or 2.5

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*Associate Professor, Clinics and Medicine, School of Veterinary Medicine, University of Georgia.

®[1]Winthrop-Stearns, Inc., New York, N. Y.

®[2]The Armour Laboratories, Chicago, Ill.

cc. of ECP[3]. This is best administered as soon as retention of placenta is recognized. Further treatment recommended includes: Nux vomica tonic, sulfonamides orally, and 2,000,000 to 3,000,000 units of penicillin intramuscularly.

Capsules containing disinfectants and deodorants may be inserted into the uterine cavity. Four to 5 oz. of tyrothricin in capsules placed in different places around the uterus serve well. Novoxil[4] works well. My favorite for the last year has been the insertion of 1 or 2 gm. of terramycin[5] in tabule form. Break tabules up well as they do not dissolve rapidly. In 24 to 48 hours placenta is passed or loosened so it may be removed easily.

A cow sick from retained placenta needs supportive treatment. One to two liters of 5% dextrose (not over 10% solution if the cow is dehydrated) may be given intravenously slowly. The commercial 40 to 50% dextrose will further dehydrate an animal already low in tissue fluids. Two or 3 oz. of mycoban[6] to cows partially off feed, given twice daily, is often worth prescribing for two or three days. Profloran[7] *via* stomach tube, combined with ferro-cobalt compound[8] or a half pint of molasses in a pail of warm water, is of value.

You can do the cow owner a real service, after the cow has made an apparent recovery, to suggest reexamination to be sure the uterus is back to normal. Much can be done to speed breeding condition by massage of the ovaries and uterus, removing ovarian cysts and the use of intrauterine infusions of penicillin or streptomycin. Aqueous solutions containing 1 gm. of streptomycin are good treatments for these cases as well as those with a few flakes of pus or a slight swelling of one horn. Treat during estrum, when the cervix is relaxed, by infusion *via* plastic insemination tube.

Acetonemia

Many cases of acetonemia are brought to our clinic for treatment every year. I am sure we average two or more cases a week in addition to those treated as ambulatory cases. If diagnosis is acetonemia we like to give 500 cc. of 40% dextrose intravenously and 2 or 3 oz. of sodium propionate or mycoban by capsule and dispense 1 lb. of mycoban to be given twice daily as a drench in 2 or 3 oz. doses in water. If the cow is nervous, 1 oz. of chloral hydrate is given. We are told that chloral releases all glycogen from the liver and, if that is true, repeated doses are no better than one.

Badly dehydrated animals are hospitalized and given repeated injections of less concentrated dextrose solutions. A good tonic containing strychnine is often used; also cobalt in the form of ferro-cobalt compound.

It is always good practice to examine the uterus by rectal palpation for evidence of metritis or retained placenta. Also look for mastitis or other infections, in which case acetonemia is probably secondary. A distinct form of this condition exists in the South. It is seen often in thin cows that are badly parasitized. In the North, it is most often observed as a disease of overfeeding or in cows that are overconditioned.

In those cases that have run for weeks, and ones that are certain to have extensive liver damage, mycoban is beneficial when given daily over a period of two weeks or more. One case of this kind was sent in recently by another veterinarian. This cow had been treated several times with dextrose during a period of three weeks. A peculiar stiff attitude was noted, and percussion demonstrated sensitivity over the liver. The animal had lost condition almost to the point of emaciation and presented a syndrome similar to traumatic indigestion. Production had dropped from 55 lb. daily to about 4 lb. Mycoban only was used as treatment. Full feeding of alfalfa hay and grain was given. The cow came back to 40 lb. of milk and gained flesh rapidly in the next two weeks. The owner advised a month later that the cow was producing 55 lb. of milk daily and was back in normal condition.

We have used a quantity of cortone[9] and hydroxycorticosterone (compound F) supplied by Merck & Co. for experimental use in acetonemia. Of 16 cases, 12 did not require further treatment after one dose of 1 gm. cortone. Two of this group required a second dose and the final outcome of the other two was not learned. These cases were all treated in the farmers' trucks and sent home. We were careful to pick uncomplicated cases, but some of these were severe. Approximately equal results follow one dose of 0.5 gm. compound F. If we had but one treatment for acetonemia, I would prefer cortone or a similar acting product. In some cases, I would combine dextrose and supporting treatment. For a one-call treatment, cortone appears to be

[3]The Upjohn Company, Kalamazoo, Mich.
[4]E. R. Squibb & Sons, New York, N. Y.
[5]Charles Pfizer Company, Brooklyn, N. Y.
[6]E. I. DuPont DeNemours and Company, Inc., Wilmington, Del.
[7]Jensen-Salsbery Laboratories, Inc., Kansas City, Mo.
[8]Pitman-Moore Co., Indianapolis, Ind.
[9]Merck Co., Inc., Rahway, N. J.

reliable, but many Georgia farmers object to the cost.

Sterility

Since the advent of artificial insemination, demand for treatment of infertility has increased. Inseminators are required to keep records that reveal how many times cows have been bred. This is information that was not available under the system of pasture breeding. Furthermore, insemination fees cause the dairyman to examine breeding records and losses occasioned by failure to get cows settled.

Many cases are observed having a history of being inseminated four or five times without conception. Such animals have to be handled according to facts of breeding history, but when an examination reveals nothing abnormal, our treatment is 1 gm. streptomycin plus 200,000 units of aqueous penicillin in 20 to 30 cc. of saline injected into the uterus *via* a plastic insemination tube. Another treatment recommended is 30 to 40 cc. of tyrothricin. When these animals are treated at the beginning of estrum, they may be bred later the same day (three to four hours later). Some prefer to breed the cow and infuse the uterus later the same day or even the next day. Vital sperm are out of the uterus and in the fallopian tubes within a matter of minutes so this can do no harm. The fertilized egg does not reach to the uterus for at least three days, so infusion after breeding may be a good way to prepare the uterus for implantation.

Another treatment we like for cows that have no recognizable pathology, but regularly come in heat without conception, is the synthetic estrogen dinovex®[10]. There is a feeling that many of these cows are slow ovulators and something is needed to stimulate the production of estrogen and speed the rupture of the follicle. Dinovex is claimed to supplement ovarian estrogen production within a few hours after injection. When injected at time of service, it stimulates ovulation while sperm are still viable. Of the first five cases in which I used this product, three conceived at first service.

Prepartum Prolapse of the Vagina

In the summer issue of *The Allied Veterinarian*, Doctor Burch reported on the use of progesterone in treating prepartum prolapse of the vagina. This serious and hard to handle condition is especially difficult to treat in beef cattle. It is often accompanied by prolapse of the rectum. Treatment suggested has been replacement of prolapsed organs and suturing the vulva to hold them in place. To prevent straining, repeated injections of epidural anesthesia must be used.

This condition may be due to excessive estrogen in many cases. Similar pathology has been produced by implants of stilbestrol. Progesterone should be the antidote for this estrogenic stimulation. The product is repositol progesterone®[11], using 1 mg. per 5 lb. body weight on recent cases and double the amount on long standing cases. With adequate dosage the straining should stop in 24 to 48 hours. In persistent cases the dose is repeated. We have had little experience with this product, but one case will illustrate: The patient was a four-year-old Angus cow, pasture bred, and approximately two weeks from calving. This cow had a severe prolapse of the vagina and a mild protrusion of the rectum. Straining was severe. A small amount of epidural anesthesia was injected, the vagina and rectum replaced and the cavity of the vagina and rectum massaged with V-C-B-U ointment®[12] for its anesthetic effect. No suturing was required. At the same time, 300 mg. of repositol progesterone were injected subcutaneously. Calving was uncomplicated seven days later.

I hope we have something here that will eliminate a large percentage of these cases without surgery or suturing. This treatment can not be expected to give satisfactory results on prolapse after calving that is due to injury or to necrotic vaginitis.

Leptospirosis

This is a disease that is presently in the limelight. It has been reported from most of the states and from many places where it had not been suspected.

The few cases presented to our clinic were acute and were referred for diagnosis. Chronic cases are the rule. Symptoms of icterus, bloody milk, hemoglobinuria, abortion, and high body temperature do not always characterize the infection. Bloody milk or a slight pinkish coloration to the milk, with no evidence of inflammatory changes, is suspicious. More often the milk is just thick, sticky, and yellow. Urine may be coffee colored. Sticky secretion from the udder of one case was suspicious of leptospirosis but urine voided was normally clear. Leptospira organisms were found in the urine.

Here are some facts brought out in a recent

®[10]Ortho Pharmaceutical Corp., Raritan, N. J.

®[11]Pitman-Moore Co., Indianapolis, Ind.

®[12]The Warren-Teed Products Co., Columbus, Ohio.

talk by Doctor Baker. There are about 20 different species involved in infections in various regions of the country but *L. pomona* is the one most often found. Infection is generally from urine absorbed through wounds and even through unbroken skin. Organisms multiply in the blood. They may be found in circulating body fluids three or four days after infection, reaching a maximum concentration in about ten days, then disappearing from the blood stream and localizing in the kidneys. Chronic cases are as potent carriers as acute cases. One bull is known to have been a carrier for six years.

Of 120 cases of periodic opthalmia in horses, all revealed infection with Leptospira. Little is known about this disease in horses. The infection appears to be mild although incidence may run as high as 20%. Another factor about leptospirosis is that it is most readily spread from cow to pig or from pig to cow but not so readily spread from cow to cow. Incidence in swine is unknown.

Many farmers with cows buy pigs from unknown sources. They should be advised against this practice because of the danger of introducing infection.

In the differential diagnosis, four important diseases need to be considered where an abortion storm breaks: Brucellosis, vibriosis, trichomoniasis, and leptospirosis. Leptospirosis must be differentiated from mastitis, pyelonephritis, and bacillary hemoglobinuria. There are cases where abortion is the only recognized symptom.

The complement fixation test is the most reliable for accurate diagnosis. Samples are taken as for brucellosis, in fact the test can be run for both from the same sample. Often only one or two cases are seen in a herd, but a Nebraska herd where 105 cows aborted in five weeks illustrates what can happen. Baker believes that if leptospirosis can be eliminated from swine it would not maintain itself in cattle. He also states that guinea pigs are not as satisfactory as hamsters as test animals.

All the antibiotics appear to be leptospirocidal but variability of symptoms precludes specific treatment. Penicillin, streptomycin, and terramycin have been recommended. We have used streptomycin on acute cases without results. In chronic cases streptomycin has relieved symptoms, but there is increasing evidence that these cases may be carriers. Baker states that the broad spectrum antibiotics may relieve the carrier state. He is certain, however, that penicillin will not do it.

A new vaccine recently made available is a chick embryo vaccine, freeze-dried under vacuum. Procedures for use include testing for brucellosis, vibriosis, and leptospirosis and vaccination of negative animals if Leptospira reactors are found in the herd. Positive animals should not be vaccinated.

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Mastitis

(*Continued from page 56*)

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For ringworm in calves mix 1 gm. of iodine crystals with the contents of one tube of brushless shaving cream and apply the mixture directly to lesions. The shaving cream vehicle is better than petrolatum. — *John D. Cady, D.V.M.*

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Inflammatory changes in the lining of the rumen may result from too rapid shift from forage to concentrated rations. These inflammatory changes are associated with liver abscesses.—*Rue Jensen, D.V.M.*

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Spermatozoa are susceptible to any but favorable influences. They have no mechanisms to protect themselves from unfavorable influences.

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Whatever treatments or combination of therapeutic efforts are used in the control of keratitis as a herd problem, such treatments should always be supplemented with increased vitamin A and riboflavin content of rations.

Nutritional Deficiency Diseases Of Animals†

THOMAS J. JONES,* B.S.A., D.V.M., M.Sc., Ph.D., Athens, Georgia

THE veterinary profession has been confronted for many years with the overall problem of animal nutrition; however, only recently have we been aware that the nutritional status of the individual animal may contribute greatly toward the success or failure of the practitioner when called upon to treat ailing stock. The prognosis in these cases reflects directly upon the ability of the attending veterinarian, and many of us have found out (too late) that we are faced with an impossible situation. In no phase of veterinary practice, in small, and especially in large, animal work, does an outside influence play such a part as the nutritional status. This is such a simple and obvious fact that we are prone to overlook it in seeking some spectacular and startling remedy. The unobtrusive and insidious nature of many nutritional factors tends to obscure the real basic cause of disease and the postmortem record will show "unknown" as the cause of death.

The source of all nutrients for plants is in the soil and the close relationship between soil fertility and the quality of forage or grains is well known. This brings us to two well known maxims, that good quality forage crops can not be expected from poor, deficient soil, and that it is impossible to starve a profit from an animal. It has been tried many, many times and will undoubtedly be tried again, either knowingly or otherwise. Classical research of Becker and associates in the late twenties and early thirties at the Florida Experimental Station, Gainsville, on cobalt and copper still stands as the key that opened the door to many nutritional problems. The veterinary profession at that time became acutely aware of the need of much information on the role of trace elements in animal nutrition. We have just lately become aware of nutritional deficiencies being important predisposing factors in other and more serious diseases of livestock and pets. Another condition with which all experienced practitioners are familiar is the association of extreme parasitism with malnutrition. Which of these conditions is primary and which secondary is still a debatable academic question. However, we find in our clinic that an emaciated and thin animal usually carries a heavy parasitic load and is frequently too weak to withstand the therapeutic dose of an acceptable anthelmintic. This is true both in large and small animal practice.

We find nutritional deficiencies resulting from deficiency of minerals, including trace elements, and vitamins. The importance of vitamin content of feed is being better understood as more information resulting from experimental data is made available.

Much data available have been gathered by experiment station investigators working independently of the veterinary profession and without the help of trained veterinary pathologists. Another source that has unraveled some of the most perplexing questions is feed producers. These institutions have supported educational institutions through grants in aid, scholarships, and other financial support, through which we, as a profession, have benefited directly by applying facts gleaned from results of experiments. The profession must stimulate a revival of interest along these lines so that results might be interpreted in the light of field experience.

Essential elements for maintaining health of animals, according to the information available at present, are as follows: Calcium, phosphorus, magnesium, sodium, potassium, chlorine, iron, copper,* cobalt,* iodine,* sulfur, manganese,* and zinc*.

Other elements present in the animal body, but not found to be essential (although excesses may be harmful), are as follows: Aluminum, silicon, fluorine, selenium, molybdenum, arsenic, and nitrates.

It is true that the young of all species of domestic animals and pets may need all of the above. It has been shown that mature animals can be supported adequately by the addition of the following: Calcium and phosphorus, salt and iodine. With the calcium and phosphorus must be an adequate amount of vitamin D.

†Presented at the Florida State Veterinary Medical Association annual meeting, Daytona Beach, Oct. 18-20, 1953.

*Dean, School of Veterinary Medicine, University of Georgia.

*Trace elements required.

Vitamin Requirement of Animals

Vitamins in livestock nutrition are divided into fat and water soluble. Mature animals usually find sufficient amounts of all vitamins in their rations, but occasionally unusual conditions may cause a deficiency of the vitamin or precursor in the ration. A striking example is a deficiency of vitamin A that sometimes appears in the early spring and again in midsummer. A deficiency of vitamin A is observed in dairy cows in the spring after being fed all winter on hays that were too mature or lacked green leaves when the hay was harvested during the preceeding summer and also while grazing on drought-parched pastures during late summer and early fall. It is observed, during such times, that calves dropped from such vitamin-depleted cows fail to survive because their supply of vitamin A is too low, or even nonexistent.

Vitamins can be classified as follows:

Vitamin A or provitamin A
Vitamin D
Vitamin E
Vitamin K

Vitamin B_1 thiamine
Vitamin B_2 riboflavin
Vitamin B_5 niacin (nicotinic acid)
Vitamin B_6 pyridoxines
Pantothenic acid
Biotin

Biotin
Other B complex vitamins
Choline
Folic acid
Paramino acid

The symptoms of deficiency of minerals and vitamins can be grouped for the purpose of this paper. A deficiency of calcium, phosphorus, and vitamin D results in rickets.

Deficiency of vitamin E results in impaired reproduction or muscular dystrophy.

A deficiency of vitamin K is seldom, if ever, seen in large animal practice. It is not uncommon in poultry practice and most reports of such deficiency have been reported by workers in that field. It can and does occur occasionally in dogs and is worthy of consideration from that standpoint. Animals that are deficient in vitamin K are poor surgical risks.

Vitamin B Deficiencies. The vitamin B complex includes all of those of the vitamin B group that are known to be needed by animals. The small animal practitioner is most directly interested in this group, except possibly the general practitioner in connection with the need of vitamin B in certain species such as swine during early life of the animal. It has been shown that deficiency of the B vitamins does occur ocassionally in pigs at about weaning age. Other species, such as puppies, definitely can be deficient in one or more of the B vitamins. From the practitioner's standpoint, it is impractical, if not impossible, to make a definite diagnosis of a deficiency of any one particular vitamin fraction, so we resort to a mixture of B complex that is available for injection. Since an excess of those not particularly needed is presumably not harmful, the entire group is injected either subcutaneously or intraperitoneally at daily intervals.

The young of all species also need three mineral elements, including copper, cobalt, and iron. Large areas of the country have sufficient amounts of these elements in the soil and, where the animals have access to the soil, their requirements may be met. However, young pigs frequently need a supplement if they are to grow as rapidly as is possible.

The above paper covers all of the presently known requirements of animals for vitamins except for vitamin C or ascorbic acid and also for vitamin P, or citrin. There may be a strong correlation between vitamin P and vitamin K, as both appear to be concerned with the vascular system. Vitamin K causes blood to clot, while vitamin P concerns the fragility of the blood capillaries. The veterinary profession needs to do a vast amount of research of the dietary needs of animals and especially of the interrelation of one dietary factor with another. The ground work has been done and we should learn that we must be able to stand on our own feet and help ourselves.

Fundamentals are known and we must channel efforts into getting trained personnel to take up the challenge of finding answers to questions posed today. We have delayed too long and depended on other allied professions to supply us with information that we use every day in veterinary practice.

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Infections Of The Bovine Tubular Genitalia†

G. R. MOORE,* D.V.M., M.S., East Lansing, Michigan

IT is the purpose of this paper to discuss briefly only those infections of the vagina, cervix, and uterus that are mild and chronic, and characterized by infertility. Accustomed as we are, as practitioners, to dealing with violent infective and inflammatory processes, we have been perhaps a little slow in recognizing in their true significance these subclinical infections of the tubular genitalia. Recent studies by Easley,[1] subsequently confirmed in our clinic, indicate that these low-grade processes are the cause of 85% or more of infertility in cattle. Considered in this light, they constitute one of the most important disease entities in the entire field of veterinary medicine.

Being warm, moist, mucus-coated, hollow organs, the vagina, cervix, and less frequently the uterus, are ideal locations for infections so mild in nature as scarcely to be recognizable, but capable of acting as effective barriers to conception.

Careful studies show that even the mildest inflammations of mucous membranes cause a marked change in the reaction of their secretions toward the alkaline side. Spermatoza are capable of surviving only in media having a narrow pH range of 7.1 to 6.4 (neutral to slightly acid). In addition, many bacteria elaborate toxins injurious to spermatozoa.

A further consideration in evaluating the effects of these mild infections is the role played by the rather abundant estrual mucus secretions. It is now fairly well established that estrual mucus in cattle is important as a vehicle and activator diluent for the extremely concentrated semen of the bull. By actual count, the average bull ejaculate of from 3 to 8 cc., contains, on the average, as many sperm as the average equine ejaculate of 75 to 150 cc.

If a drop of live bovine semen is placed on a microscope slide in close proximity to a drop of normal estrual mucus, and the two are teased together while under microscopic observation it is observed that the spermatozoa are stimulated by contact with the estrual mucus. Motility increases and the individual sperm penetrates rapidly through the mucus. When the experiment is repeated, using estrual mucus from a cow with vaginitis and cervicitis, motility drops off quickly and the sperm soon die. In the past, we used this phenomenon as a laboratory demonstration and now use it as a simple effective test for subclinical infections that may prevent conception.

Where infection is mild and limited to the uterus, the above test may not work unless the mucus is obtained directly from the uterus by catheterization and aspiration. A Nielsen-Frohner uterine catheter is favored for obtaining these samples.

If one does not care to make this test, or lacks the equipment, it is reasonably sound to make a diagnosis by playing the law of averages. It is now well established that at least 90% of those cases of infertility in cows with normal estrual cycles, and in which no pathology can be recognized, are due to these low-grade infections.

These cases usually can be cleared up by one or two treatments. Using a Chambers or Nielsen-Frohner cervical catheter, we inject into the uterus 300,000 units of crystaline penicillin and 1 to 2 gm. of streptomycin, using as a vehicle 40 cc. of a commercial solution of one of the sodium salts of sulfonamides. This procedure can best be accomplished during estrum. Half of the antibiotic is deposited deep in each horn. The cow is bred at the next heat period following treatment. Sixty-five percent of our cases thus treated have conceived on the first service.

Catarrhal vaginitis is observed frequently as a cause of infertility. Occasionally its occurrence is widespread in a particular herd causing breeding failure in a majority of the females. Examination reveals a catarrhal exudate in the vault of the vagina and the mucosa presents a dirty, brick red appearance. Strands of grayish colored exudate frequently are seen stretched across the dilated vaginal walls.

This condition is usually the result of repeated contamination of the vagina with coliform and similar organisms. It is common in big, fat, or old cows in which relaxation of the vulva allows exposure of the vaginal mucosa during recumbency, or in cows where a high

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*Director, Large Animal Clinic, Michigan State College.

tailhead and receding anus cause tilting of the vulva. In these cases, we do a modified Caslick's operation to close the upper half or two-thirds of the vulva, using the method recently described by Roberts[8].

In practically all cases where catarrhal vaginitis is widespread in a herd, diligent search has revealed a common source of repeated contamination, such as dirty methods of artificial insemination, and bulls in which filthy environment has caused matting of the preputial hair resulting in pus producing infection of the preputial orifice. In one herd, sadism on the part of an attendant was finally found to be the cause. In still another herd, hosing down the cows at milking time was demonstrated to be the basis of a widespread catarrhal vaginitis of a severe type. A large hose with a fire type nozzle was being used; the idea being thus to gain enough pressure literally to knock the dirt off the cows. Vaginal examination following its use revealed that fecal material from around the tail head was being forced through the vulva, in some cases as far as the cervix.

In these "epidemics" of vaginitis it is, of course, imperative to seek out and remove the cause. This may be easy or extremely difficult, but no measure of success in controlling the condition will be attained until this has been accomplished. Individuals respond well to treatment which consists of medicating the vagina with a mild, protective, long acting, antiseptic preparation. A great many products fit these requirements. Our standard treatment is to insufflate the vagina with bismuth-formic-iodide healing powder. Two or three treatments at three-day intervals are usually sufficient. In applying powders to the vagina with an insufflator, a speculum should not be used. If the nozzle is inserted and the vulva pressed tight around it to prevent the escape of air, ballooning occurs during the procedure, thus allowing for a much more uniform application of the powder than is otherwise possible.

Granular vaginitis is a chronic venereal infection of cattle and is usually considered only when cows that are apparently normal in all other respects fail to conceive when bred. There are granular or nodular elevations on the walls of the vulva and vagina which can best be seen under a strong light.

Elevations can be found in practically all cows, and it should not be assumed that the presence of a few such nodules will explain poor breeding efficiency.

The typical nodules in a chronic case of granular vaginitis are colorless, or have a faint yellowish center surrounded by a vascular girdle, and are noted only on the posterior one-fourth of the vaginal mucosa. This condition is greatly aggravated by copulation. Within 24 hours after service there is marked irritation; the mucosa is scarlet, swollen, and tender. Many cases show a discharge which is a mixture of varying amounts of mucus and pus. The nodules now multiply rapidly and take on an angry red color. In a few days the acute symptoms recede.

If the cow conceives at the first service, pregnancy may proceed normally. What is more likely is failure to conceive, and at the next service the condition is aggravated further. The most typical lesions are found in heifers that have been bred and rebred many times. In these, such infection is an effective barrier to conception.

When a cow with granular vaginitis fails to conceive after three breedings, it is advisable to withhold from service for one or two estrual periods and treat the condition. Insufflation with bismuth-formic-iodide powder at weekly intervals is usually followed by satisfactory results. Painting the affected areas with a saturated aqueous solution of gentian violet once a week for three weeks is another excellent method of treating granular vaginitis.

Mild chronic pyogenic endometritis or *pyometra* is a fairly common cause of infertility. It follows retention of fetal membranes, abortions, abbreviated pregnancies, and occurs occasionally in virtually every cow in a herd where an impotent sire is in service. In this latter type, it is due to early death of improperly fertilized ova. Dead embryos that are not expelled attract infection from every available source. Persistence of the corpus luteum of pregnancy favors retention of the exudate, thus establishing a chronic pyometra.

The accurate diagnosis of chronic pyometra demands an authentic history, and a detailed clinical study. Vaginal examination usually reveals small quantities of pus exuding from a relaxed cervix. On palpation per rectum, the uterus is found to be distended with pus so as to simulate quite closely early pregnancy.

If a cow has suffered from retained afterbirth the animal may have lost, by necrosis, all caruncles from one or both horns, in which case there is small chance that she will ever again produce a live calf. This type of damage is not usually recognized at the time it occurs. After the condition has resolved to a pyometra the presence or absence of caruncles can not

be determined by clinical examination. Consequently, the prognosis in pyometra is always guarded.

For the past ten years, I have made it a practice to follow the manual removal of retained afterbirth with the administration of a suitable dose of stilbestrol, or other estrogen, in all valuable breeding females. This treatment does the most good when given from 10 to 14 days after cleaning. By this time all remnants of afterbirth and other debris have liquified and the estrogen-induced heat relaxes the cervix, increases uterine tone and blood supply. and causes rhythmical contractions of the uterus effectively unloading the uterine contents. This procedure greatly reduces the incidence of chronic metritis and results in preserving the fertility of a large percentage of these cows.

In treating chronic pyogenic endometritis, a chief problem is gaining entrance through the cervix. If the cow is coming in heat regularly, it is best to wait for relaxation of estrum. In most cases, however, foreign material in the uterus causes retention of the corpus luteum and the cow fails to come in heat. A dose of 10 cc. of estradiol cyclopropionate (ECP)® will accomplish this in 24 to 48 hours. We prefer ECP to stilbestrol for this purpose, because of its more persistent action.

When the cow is in heat, the uterus can readily be douched. A pair of Knowles cervical forceps is anchored to the lip of the cervix, which is then pulled to the posterior part of the vagina. With his arm in the vagina the operator can then grasp the cervix and guide a return flow catheter into the uterus. A gallon of water, to which an ounce of salt has been added, is as good as any other douche. Gravity flow is used from an elevated douche bag connected by rubber tubing to the catheter. I introduce 100 to 300 cc. of saline at one time, and with one hand in the rectum, I massage the uterus to dissolve the pus in the saline and then allow it to escape through the return flow. The procedure is repeated until the return flow is clear. After all fluid is carefully removed, a generous amount of penicillin and streptomycin is introduced and left in the uterus. In some cases it is necessary to repeat the treatment after two or three weeks. Affected cows should not be bred for at least three heat periods after recovery.

®The Upjohn Company, Kalamazoo, Mich.

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Short Subjects and Clinical Briefs*

Interpretation of the Tuberculin Test

In order to interpret correctly the intradermal tuberculin test, it must be ascertained that injection was made properly. Three essentials of technic are required: (1) Inject the diagnostic agent properly, (2) provide adequate restraint of animals, and (3) use recommended quantity of tuberculin. If these items are accomplished with exacting care, the second step, observation of reaction, should be simple, keeping in mind individual herd histories, additions, and clinical manifestations, if any.

Several factors may influence reactions to various degrees:

1. Sensitivity of individual animals to tuberculin differs. This is noted frequently in young animals on first test.

2. Variation in thickness of the skin may influence size of reaction and will vary depending on the body area used.

3. One must be mindful that a desensitized area develops, extending for three to four inches from the point of injection. One must allow sufficient time between injections if the same test area is to be used.

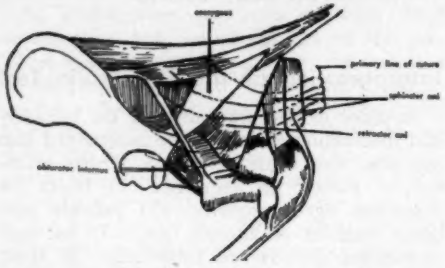
4. Consideration must also be given to possible physiological reasons that might influence reactions, such as recent use of biological products. It is well to inquire if any animal is under treatment of any kind.

Differentiation between suspicious reactions, in order to classify them as positive or negative, may be possible by following one of three procedures: (1) Make a 96- to 120-hour rereading on the original injection, (2) reinject in a neutral area and observe at the prescribed time, or (3) inject 5 to 10 cc. of intradermic tuberculin subcutaneously, preferably in the prescapular area, then observe reaction closely for six to eight hours. If the animal is tuberculous, reaction will increase in size.—*P. W. Wassenaar, D.V.M.*

*Presented at the Florida State Veterinary Medical Association annual meeting, Daytona Beach, Oct. 18-20, 1953.

Anatomical Factors in Perineal Hernia

Recurring perineal hernia after surgical repair and castration may result from failure to give proper consideration to anatomical structures involved. Many unfavorable results may be attributed to a lack of knowledge of



the anatomy of the region and improper placing of the initial line of supporting sutures.

When carefully explored, three primary muscles and their fascia enter into repair of hernia in the perineal regions. These are the coccygeus, obturator, and sphincter ani with fascia of the gluteal region. Accompanying diagram indicates the location of the primary line of sutures.—*Frank Meuller, D.V.M.*

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Hench-Aldrich Test for Blood Urea

Mix 5 cc. of fresh blood with 5 cc. of 10% trichloracetic acid. Centifuge to remove protein. This produces a chocolate-colored sediment and clear supernatant fluid. It will take about 30 minutes to obtain 5 cc. of clear fluid.

Fill 30 cc. burette with 5% bichloride solution. Run into a tube with the 5 cc. of clear fluid, 2 cc. of the bichloride solution.

Add 0.25 to 0.5 cc. of bichloride solution and shake or stir well. Then add 0.25 to 0.5 cc. mixture to a drop of a saturated sodium carbonate solution on a spot plate or white piece of glass or china.

Keep repeating the drops and testing until the end point is reached, which is indicated by a brick red color.

When near the end point, sodium carbonate solution will begin to show canary, which increases in intensity when near the end point. From this point, begin to count to three and, when the end point is reached, it will have turned brick red (three seconds).

Multiply number of cubic centimeters used from bichloride solution by 40; from this subtract 60 and the difference will express the number of milligrams of urea in 100 cc. blood. To get blood urea nitrogen take 46.65% of urea.

Blood urea of normal dogs ranges from 20 to 40 mg. Animals infected with leptospirosis or nephritis will give readings from 250 to 400 mg.; uremia, 360 to 975 mg.—*E. F. Thomas, D.V.M.*

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Rapid Leukocyte Determination

The method as described is used only on out patients or large animals where only one trip will be made. It has not been designed to replace the standard blood counting technic.

Materials. Standard syringes, small gauge needles, 2 or 3% glacial acetic acid, microscope and counting chamber.

Method. (1) Prepare clean equipment. (2) Draw 0.1 cc. of blood in a 2 cc. syringe and immediately fill to 2.1 cc. with the acid. (3) Shake the syringe, discard few drops and flood the counting chamber. (4) Count the four corner squares and multiply by 50.

Discussion. In my experience, the deviation of error with the standard technic is no more than 15%. Increased strength of acid produces more rapid and complete hemolysis of the red cells. For convenience, keep diluent in rubber stoppered vials. A 5 cc. syringe may be used, diluting 0.1 cc. of blood to 4.1 cc. and using a factor of 100. Theoretically, needles should be changed before dilution, but if a 20- or 22-gauge needle is used error is not great. The total leukocyte count alone should not be overrated in making a clinical diagnosis.—*Wm. F. Jackson, D.V.M., M.S.*

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Treatment of Granular Vaginitis

A powder consisting of a mixture of silver picrate 1% with kaolin *q.s.* is used in our practice for the treatment of granular vaginitis in cows. Application is by use of an insufflator. Treatment is repeated at five- to seven-day intervals for two or three treatments. Occasionally, slight discomfort of the patient is noted following administration and some exfoliation of the mucous membrane may be expected. Breeding rest of from 60 to 90 days is recommended.—*F. W. Brown, D.V.M.*

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A handy pack for autoclaving cotton suture may be made from a short piece of plastic tube. Suture is wrapped around the tube and the free end fastened in a notch at the end of the tube.—*J. O. Knowles, V.M.D.*

Ethyl Chloride as a General Anesthesia in Puppies and Cats

A small piece of gauze or a surgical sponge is held, with forceps, well back in the oral cavity and ethyl chloride applied directly to it. Anesthesia is almost instantaneous and lasts for only a brief three to five minutes. This is sufficient time for minor surgery such as castration in the tom cat, tail or dew-claw amputation in puppies.—*C. Paul Vickers, D.V.M.*

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Catheterization of Small Bitches

The first consideration for ease in catheterizing female dogs is their proper restraint. Illustrated is a most satisfactory method for small females. After insertion of a catheter

having a small lumen, attach a sterile glass syringe and withdraw quantity of urine desired.

Sterilization of catheters is an important consideration to avoid contamination of the bladder and resulting cystitis.—*Mark L. Morris, D. V. M.*

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Use hydrocortisone ophthalmic drops in the treatment of non-specific keratitis and conjunctivitis.—*R. F. Minnick, D.V.M.*

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A drop or two of butyn in the nose of cats facilitates passage of a nasal catheter.—*Harlan Jensen, D.V.M.*

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Obtundia (Clapp, Otis) is excellent to keep dogs from licking wounds.—*C. Paul Vickers, D.V.M.*

A length of 1-in. adhesive tape, looped around the right or left front or rear leg just above the paw, and continued to include the opposite leg in a second and third loop, if desired, provides good restraint of cats.—*Harlan Jensen, D.V.M.*

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A powder mixture of streptomycin and penicillin is excellent for therapy of open wounds. —*Ivan Fredrickson, D.V.M.*

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Sprinkle salt on the tongue and lips of dogs that have been off feed or vomiting, to replace the HCl lost and prepare the stomach for food.—*Mark L. Morris, D.V.M.*

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Treatment for Chronic Fungoid Dermatitis

Salicylic Acid	1 oz.
Tannic Acid	2 oz.
Roccal	1 oz.
Alcohol 70% *q.s. ad.*	1 gal.

Sig: Apply to infested skin once daily.—*Albert Knowles, D.V.M.*

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One-quarter dose of nembutal (Abbott) mixed with pentothal increases the duration of anesthesia and reduces the quantity of pentothal needed for short periods of anesthesia. It is a safe anesthetic.—*Stanley Wasman, D.V.M.*

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A mixture of ether and oxygen is the safest anesthesia for poor risk surgical patients.—*Robert P. Knowles, D.V.M.*

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Treatment for Bacterial Ear Infections

Mix contents of 100 mg. aureomycin® capsule in ½ oz. of sulfadiazine powder. Dust in ear t.i.d.—*Fred Thomas, D.V.M.*

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Traction for reduction of fracture of the radius and ulna in a dog by applying the body weight of the animal to the fractured limb assists the operator in applying a cast.—*Ronald Jackson, D.V.M.*

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Carbon dioxide 5% and oxygen 95% inhalation is of great help in neonatal asphyxia. —*J. O. Knowles, V.M.D.*

® Lederle Laboratory Division, Pearl River, N. Y.

In cases of difficult calf scours, give 250 mg. terramycin[®1], b.i.d. for six doses.—*W. C. Holland, D.V.M.*

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For insemination of cows I like to deposit semen deep into the uterine horn.—*J. E. B. Mouw, D.V.M.*

▾ ▾ ▾

In treatment of uncomplicated swine pneumonia give all hogs:

5 cc. mixed porcine bacterin, no. 1,

1 gr. per lb. of body weight sulfamerazine, and hog cholera antiserum.—*W. R. Brawner, D.V.M.*

▾ ▾ ▾

Modification of Caslick's operation, suturing the upper commissure of the vulva, has been used with success in some cows for the relief of vaginitis. Metal suture clips are used as recommended for the operation in mares.—*W. R. Brawner, D.V.M.*

▾ ▾ ▾

Treatment of Bacterial Infection of Swine

Penicillin in oil	1,000,000 to 2,000,000 units
Streptomycin	1 to 2 gm.

Inject separately

—*T. W. Brown, D.V.M.*

▾ ▾ ▾

Treatment for Bovine Metritis

Inject the following mixture into each uterine horn:

Neoprontisil	50 cc.
Water	50 cc.
Streptomycin	5 gm.
Penicillin	1,000,000 to 2,000,000 units

—*J. E. B. Mouw, D.V.M.*

▾ ▾ ▾

Amino acid powders fed to pups, after deworming for hookworms, will assist in protection from reinfection. — *Ronald Jackson, D.V.M.*

▾ ▾ ▾

A new barbiturate, kemithal[®2] has the advantage of safety, no excitement on recovery after administration and surgery, and non-irritation to subcutaneous tissue. It is classified as a short-acting intravenous anesthetic.—*George T. Edds, D.V.M., Ph.D.*

[®1]Chas. Pfizer & Company, Brooklyn, N. Y.
[®2]Ft. Dodge Laboratories, Ft. Dodge, Ia.

Castration of Tomcats Under Local Anesthesia

Inject a suitable local anesthetic, via 25-gauge needle, into the skin and tunica vaginalis. Pluck hair from the incision site with the thumb and forefinger, then clean and disinfect the area.

In young tomcats of three to four months of age, incision is made through the skin only. The entire tunic containing the testicle is grasped with mouse-tooth forceps and severed by a sudden jerk; repeat on the opposite side.

In cases of older males (six to eight months of age), incision is carried through the skin and tunic and the testicles removed by grasping at the adjacent cord and severing as above. Tunic is removed with scissors.

In old tomcats, incision is made over the testicle and through the tunic. The spermatic cord is retracted and ligated, then separated with scissors or scalpel. Excessive tunic tissue is removed with a scissors. No aftercare is recommended in any age patient.—*Wm. B. Martin, Jr., D.V.M.*

▾ ▾ ▾

Pink Eye in Cattle

Treat eye with chloromycetin[®3] and suture lids for three days.—*W. F. Jackson, D.V.M.*

▾ ▾ ▾

State meat inspection activities in Florida were conducted in some 200 slaughtering and processing establishments in 1953. Slaughtered under Board supervision were 160,545 cattle, 17,628 calves, and 511,135 hogs.—*C. L. Campbell, state veterinarian.*

▾ ▾ ▾

Treatment for Internal Parasites of Cattle

Mix 1% copper, 2% phenothiazine and enough citrus meal to assure intake ½ oz. per day in any good mineral powder. —*C. W. Kidder, D.V.M.*

▾ ▾ ▾

Louisiana boasts of the Catahoula Hog Dog, a breed of unknown origin that is doing a fine job of rounding up far-ranging hogs in the swamplands.—*Gaines Dog Research Center.*

▾ ▾ ▾

Dog-faced baboons were not susceptible to California strains of Newcastle disease virus injected intracerebrally and intraperitoneally.

[®3]Parke-Davis & Co., Detroit, Mich.

Treatment Of Ketosis In Dairy Cattle: A Clinical Report

J. L. McAULIFF, D.V.M., W. V. PHILLIPS, D.V.M., and JOHN R. STEELE, D.V.M., Courtland, New York

BECAUSE the ketotic syndrome represents a world-wide problem to cattlemen, it has claimed the attention of numerous investigators during the past several decades. Extensive work has been done, both in the field and the laboratory, to determine the etiology of the condition and the most adequate form of treatment. The nature of pathological processes and disease pathways involved has been obscure, and therapy, therefore, has been mainly aimed at alleviation of symptoms.

Interest in the postulation by Hutyra and Marek,[1] that an adrenal deficiency might be the etiological factor in ketosis, stimulated Shaw and his coworkers[2,3] to approach the problem from the standpoint of hormonal imbalance. On the basis of histopathological studies, they predicated the belief that pituitary exhaustion resulting from extreme stress of the gland at parturition causes an adrenal cortical insufficiency. That the pituitary-adrenal axis is involved was further emphasized by Puntriano[4,5].

Almost all the adrenal cortical hormones have been tried on an experimental therapeutic basis, but few have withstood the test of time. This present paper deals with our experience in treating dairy cattle, which are diagnosed as ketotics, with adrenomone®.

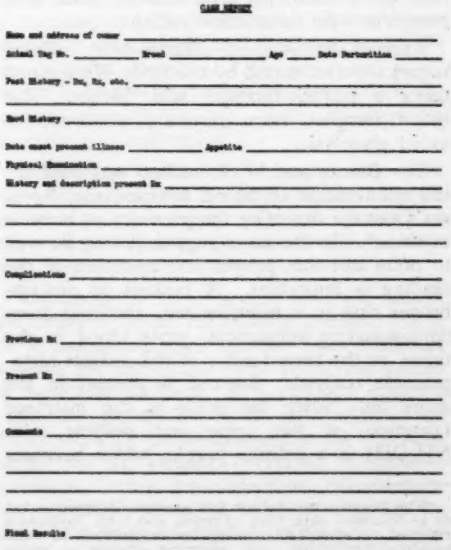
Methods

The 45 cows treated in this series were diagnosed as: (1) animals with uncomplicated primary ketosis, or (2) animals in which ketosis was accompanied by, or was secondary to, other complicating pathological processes. The cattle represented a fair cross-section of the bovine population in upper New York state, and both purebred and mixed herds were sampled. A record was maintained of temperature, respiration, and pulse rates. Biochemical assays of blood glucose and blood and urine ketones were run in selected cases. Milk production levels in pounds-per-day were watched closely.

A diagnosis of ketosis was made on cows when the physical, clinical, and clinical pathological findings revealed evidence of wasting, decreased milk production, hypoglycemia, and hyperketonemia.

Method of Recording Findings. Special case report forms were prepared by us for this experiment. Figure 1 shows a specimen chart on which are recorded the past and present history of the animal, herd history, dates of parturition and onset of present illness, space for notations as to appetite, concurrent complicating pathology, treatments previous to institution of adrenomone therapy, schedule of procedures followed in treating the present condition, comments, and final results. These results were classified as follows: (1) Recovery, (2) improvement, and (3) no improvement.

A second chart which is reproduced in figure 2 is essentially a laboratory report form, but includes space for recording milk produc-


CASE REPORT

Name and address of owner _____

Animal Tag No. _____ Breed _____ Age ____ Date Parturition _____

Past History - No, No, etc. _____

Herd History _____

Date onset present illness _____ Appetite _____

Physical Examination _____

History and description present No _____

Complications _____

Previous Rx _____

Present Rx _____

Comments _____

Final Results _____

Fig. 1

[®]The Armour Veterinary Laboratories. Chicago, Ill.

tion levels, and dates and types of treatment.

Physical Examination. Each cow was given a thorough physical examination. Graphs were prepared on body weight changes prior to and during treatment. The eye, in particular, was examined for pupillary reflex. This neurological sign is of some importance in differentiating clinically similar cases of ketosis and milk fever. There is a normal pupillary

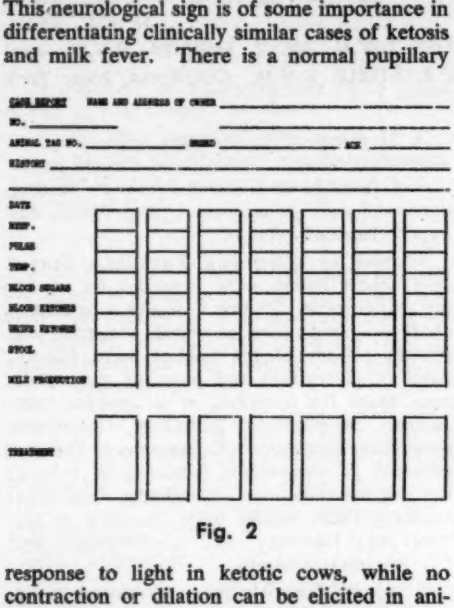


Fig. 2

response to light in ketotic cows, while no contraction or dilation can be elicited in animals suffering from milk fever*. Respiratory rate was determined by flank movement, pulse rate by stethoscopic examination, and temperatures were determined rectally.

Clinical Pathological Examination. Blood sugars were estimated by the Folin-Wu method using a sulfuric-tungstic acid filtrate. The blood samples were carefully preserved to avoid glycolysis.

The Barnes and Wick method was used for the quantitation of blood ketones, and Rothera's test for detecting the presence of acetone in urine*. In the latter regard, it may be well to point out that proper interpretation of the reading is important. A pinkish or pinkish-brown ring is a negative test, resulting from contaminating substances, gross blood in the urine, or the brown color of the sodium nitroprusside reagent. Acetone is present in the urine only when the color at the interface (junction of the urine and reagent with NH_4OH) is a definite purple, which becomes darker on standing. If the tube is then shaken, the purple color remains.

Stools were examined for consistency, and reported as normal, dry, fluid, mucoid, hard, etc. The presence of dry, mucus-covered feces is frequently noted in ketosis.

Past milking performance was studied in relation to total quantities given during the present disease. One of the first signs in the ketotic state is a drop in output, some animals even giving as little as one quart per day.

Results

This study includes records on 45 cows diagnosed as either simple or complex ketotics. The experiment was conducted over a period of one year, and was designed to measure the efficacy of adrenomone in treatment of the disease. Simple cases were limited to cows presenting a primary ketosis; those presenting one or more accompanying pathological conditions are referred to as complex.

Simple. For our purposes, we made a diagnosis of ketosis in simple cases when, in addition to positive clinical evidence, the blood sugar was less than 40 mg. %, and blood ketones were in excess of 10 mg. %. Samples were withdrawn with the animal resting as quietly as possible so that there would be little or no increase in the glucose level due to the effect of excitement on the adrenal medulla.

Table 1 shows a series of six cows (cases no. 1 to 6) with simple ketosis on which blood chemical workups were completed. Cases 1, 2, 3, 5, and 6 illustrate the usual experience in treating this type. At the onset of the experiment, we gave an initial dose of 600 A.V.U. (Armour Veterinary Units)* adrenomone, followed by 300 units at intervals until recovery was effected. Results were comparable, however, when we later decreased this to an original injection of 300 units followed by 100 A.V.U. as necessary.

It will be noticed in some cases that an animal may be classified as having recovered although the blood sugar or ketone levels may not have returned to normal. When there was a cessation of ketotic symptoms, a return to previous milk production levels and a negative urine acetone, we discontinued the laboratory work. In case 1, the cow progressed from an original blood ketone reading of 59 mg. to 7 mg. % on the 12th day, although the blood sugar showed an increase of only 1 mg. (from 37 to 38) by the same date. Subse-

*The technic we follow for acetone determination is as follows: Mix 1 cc. of urine and 1 cc. Rothera's reagent. Carefully overlay a dropperful of ammonium hydroxide, and examine against a white background for the appearance of a purple ring which indicates the presence of acetone.

*Each Armour veterinary unit provided a physiological response equivalent to 1 U.S.P. unit or 1 international unit.

quent observations indicated that all values were within normal limits.

Complex. All animals in this category had complicating pathology in addition to ketosis. Numerous investigators[7,8,9,10] have observed a variety of diseases that may precede or accompany the ketotic syndrome. Successful outcome in these cases, therefore, depends on early recognition, accurate diagnosis, and adequate treatment of all the existing abnormal conditions.

Mastitis, metritis, and traumatic gastritis provide diagnostic pitfalls for the unwary, in that the clinical appearance of the patient resembles that seen in ketosis. In addition, it must be remembered that each of these conditions may be seen during the ketotic state, and recognition requires diagnostic acumen.

Of 39 cows (cases no. 7 to 45) exhibiting complicated ketosis, 17, or 43%, had well-developed metritis; two animals, or 4%, had mastitis; and eight cases, or 18%, presented evidence of milk fever. For metritis and mastitis, antibiotic therapy was used as an adjunct to adrenomone.

In cases 40, 41, 42, 43, and 45, intravenous injections of glucose were administered for several days with little or no effect. Blood sugar concentrations rose to normal limits, but returned to previous low levels within two hours. A single dose of adrenomone was then given to each animal. Sugar and ketone values returned to normal and remained constant, evidently signifying restoration of deficient adrenal cortical factors rather than a temporary boost in carbohydrate. With the decrease in blood ketone, urine acetone disappeared.

Milk production showed an initial fall, followed quickly by return to preketotic volume.

The cows became alert, neurological signs seen in cases 5 and 22 became negative, and there was return of normal appetite with concomitant weight increase.

Dosage schedules varied in the complex cases. Most animals received 300-600 A.V.U. initially; however, one case recovered after one 200-unit injection. Cases 12 and 25 received as much as 800 units, the largest single dose given in this series. Case 12, evidently because of a severe infection resulting from a difficult retained placenta, made a slow recovery, necessitating an additional 200 units on the seventh day. Case 13 required three injections totaling 1,400 A.V.U. adrenomone, the last dose being given on the 19th day following onset of symptoms, with a complete recovery after this period. In about one-third of all cases, the single dose of 300 to 600 A.V.U. was adequate to bring about remission of symptoms.

Discussion

A review of the literature on ketosis covering the past 30 years would demonstrate the tremendous number of therapeutic agents which have been used in an attempt to correct the syndrome. Very few have proved efficacious, and some among these have been the subject of controversy between investigators. It may well be that there is no generally accepted view as to the diagnostic boundaries of the disease. Ketosis is frequently complicated by other disease entities which often mask the condition, causing it to be overlooked during the clinical work-up. This may result in recognition and adequate therapy for the ketosis, for example, but if metritis is present and not concurrently treated, the animal remains sick. Successful therapy, then, depends on an accurate and complete diagnosis.

During the postpartum period, the entire body, particularly the endocrine system, is subject to severe stress. The anterior pituitary is called on to produce increasing quantities of hormones in accordance with requirements for lactation. Heavy demands for glucose may result in pituitary exhaustion leading to a decrease in adrenal cortical secretion, consequent disturbance in carbohydrate metabolism, and a decrease in milk production. In the hypoglycemic state which results, the animal calls on its body protein and fat stores in an attempt to restore the *milieu interieur*. This excessive burning of fats results in hyperketonemia and acetonuria.

Bearing in mind this summary of events, we can understand the physiological changes in ketosis, and recognize a "classic quartet" of symptoms: Wasting, decreased milk production, hypoglycemia, and hyperketonemia.

Development of a severe infection places an undue stress on the entire organism. There is a mobilization of all the body defenses against the pathology. The endocrine system is especially affected. If a retained placenta following parturition leads to a marked inflammation of the endometrium, the resultant strain on an already overtaxed hypophysis causes a further departure from normal with the secondary onset of ketosis. The ketotic syndrome may, therefore, be seen in about 40% of all cows suffering from infectious processes.

TABLE 1.

Case No.	Owner	Animal No.	Init. dos. Adrenomone A.V.U.*	Repeat dos. A.V.U.* days	Blood Sugar Init. mg.	Blood Sugar Fin. mg.	Blood Ketone Init. mg.	Blood Ketone Fin. mg.	Urine Ketone Initial	Urine Ketone Final	Diagnosis and comment	Results
1	H. R.	21B 3584	600	300-4th 300-12th	37	38	59	7	++++	neg.	Ketosis	Rec.[1]
2	M. C.	70352	300	100-3rd 100-10th	14	33	41	10	+++	neg.	Ketosis	Rec.
3	B. V.	159	300	100-3rd	19	45	19	6	++++	neg.	Ketosis	Rec.
4	R. S.	75	300	100-3rd	37	50	16	2	++++	neg.	Ketosis	Imp.[2]
5	F. G.	VII	600	300-3rd	19	38	23	6	++++	neg.	Ketosis. Neurologic symptoms relieved following adrenomone. Milk production from 3 to 50 lb.	Rec.
6	B. W.	8760	300	100-3rd	26	45	10	1	++++	neg.	Ketosis. Milk production increased 20 lb. by 3d day therapy	Rec.
7	M. C.	64356	600	300-4th 200-8th	43	—	6	—	—	—	Ketosis, metritis, pneumonia. Dextrose, calcium penicillin	Imp.
8	S. P.	III	600	300-2nd 300-4th	248	50	55	9	++++	neg.	Ketosis, metritis. Milk production from 2 to 20 lb. 4th day. Penicillin	Rec.
9	L. L.	41105 V3	300	100-3rd	37	55	16	5	++++	neg.	Ketosis, milk fever, retained placenta, metritis. Previous dextrose therapy of no value	Imp.
10	O. C.	2644	300	100-4th 100-7th 100-9th	21	56	37	35	++++	neg.	Ketosis, metritis. Milk output from 16 to 35 lb. Dextrose, penicillin	Imp.
11	H. R.	22	600	200-3rd	—	—	—	—	—	—	Severe ketosis, metritis. Dextrose, penicillin. Milk production 0 to 70 lb.	Marked imp.
12	H. R.	17	800	200-7th	—	—	—	—	—	—	Ketosis, metritis, retained placenta. Penicillin, dextrose	Slow rec. (trouble mainly bacterial)
13	McD.	379	600	600-16th 200-19th	—	—	—	—	++++	neg.	Ketosis, metritis, retained placenta. Penicillin, dextrose	Rec.
14	McD.	412	600	200-3rd	—	—	—	—	—	—	Ketosis	Rec.
15	McD.	26	200	100-3rd	—	—	—	—	—	—	Ketosis, metritis. Penicillin	Rec.
16	McD.	52	600	100-3rd	—	—	—	—	—	—	Ketosis, septic mastitis. Dextrose, cortisone	Rec.
17	McD.	16	600	200-3rd	—	—	—	—	++++	neg.	Ketosis, metritis. Dextrose given with no improvement. Penicillin	Rec. (Fol. ACTH)
18	McD.	446	600	— —	—	—	—	—	—	—	Ketosis, metritis, retained placenta. Penicillin	Rec.

TABLE 1. (Continued)

Case No.	Owner	Animal No.	Init. dos. Adrenomone A.V.U.*	Repeat dos. A.V.U.* days	Blood Sugar Init. mg.	Blood Sugar Fin. mg.	Blood Ketone Init. mg.	Blood Ketone Fin. mg.	Urine Ketone Initial	Urine Ketone Final	Diagnosis and comment	Results
19	McD.	318	300	100-3rd	—	—	—	—	++++	neg.	Ketosis, severe neurologic symptoms. Dextrose chloral hydrate	Rec.
20	McD.	231	400	— —	—	—	—	—	++++	neg.	Ketosis, milk fever, metritis. Calcium, terramycin	Rec.
21	McD.	150	400	— —	—	—	—	—	—	—	Ketosis. Down; couldn't get up. Responded to 3-500 cc. injections—none to 4th. Complete recovery with one ACTH injection	Rec.
22	McD.	318	400	400-3rd	—	—	—	—	—	—	Ketosis, milk fever with neurological symptoms. Chloral hydrate	Rec.
23	R. D.	VI 1	300	— —	—	—	—	—	++	neg.	Ketosis	Imp.
24	R. D.	VI 2	300	— —	—	—	—	—	++	neg.	Ketosis	Good resp.[a]
25	R. D.	1400	800	200-3rd	—	—	—	—	—	—	Ketosis, milk fever, retained placenta. Milk production went from 1 to 70 lb. five days following therapy	Very good resp.
26	H. A.	V	400	— —	—	—	—	—	—	—	Ketosis, pneumonia. Penicillin	Imp.
27	O. C.	61897	400	— —	—	—	—	—	+++	neg.	Ketosis	Rec.
28	A. S.	76720	400	320-3rd	—	—	—	—	++++	neg.	Ketosis. Milk output up 10 lb. in 3 days	Imp.
29	A. S.	7304	400	200-3rd	—	—	—	—	++++	neg.	Ketosis. Milk output up 20 lb. in 3 days	Imp.
30	R. W.	97059	400	200-4th	—	—	—	—	++++	+++	Ketosis	Imp.
31	C. A.	13036	400	200-3rd	—	—	—	—	++++	neg.	Ketosis, severe milk fever. Hard, labored breathing. Calcium glucose, penicillin. Milk output from 0 to 45 lb. in 3 days	Complete rec.
32	McD.	Coquette	400	— —	—	—	—	—	—	—	Ketosis, metritis, milk fever. Calcium	Rec.
33	G. T.	?	400	— —	—	—	—	—	—	—	Ketosis, metritis. Down; could not get up. Milk output 0 to 70 lb. in 3 days. Calcium, penicillin, streptomycin	Rec.
34	G. T.	?	400	— —	—	—	—	—	—	—	Ketosis, mastitis, metritis. Milk output from 0 to 60 lb. in 3 days. Calcium, penicillin	Rec.
35	S. S.	46	600	— —	—	—	—	—	++++	neg.	Ketosis. 1 inj. glucose	Excell. rec.

TABLE 1. (Continued)

Case No.	Owner	Animal No.	Init. dos. Adrenomone A.V.U.*	Repeat dos. A.V.U.* days	Blood Sugar Init. mg.	Blood Sugar Fin. mg.	Blood Ketone Init. mg.	Blood Ketone Fin. mg.	Urine Ketone Initial	Urine Ketone Final	Diagnosis and comment	Results
36	R. W.	12098	600	600-3rd	—	—	—	—	++++	neg.	Ketosis, milk fever. Cord injury with paralysis. Calcium, terramycin	Rec.
37	A. S.	?	600	— —	—	—	—	—	++++	neg.	Ketosis	Rec.
38	A. S.	?	600	— —	—	—	—	—	++++	neg.	Ketosis	Rec.
39	C. S.	?	400	— —	—	—	—	—	++++	neg.	Ketosis, bloat, foreign body abscess. Penicillin	Rec.
40	W. B.	74192Y5	200	— —	—	—	—	—	++++	+	Ketosis, metritis, retained placenta. No improvement with previous dextrose administration	Rec.
41	W. B.	95413Y4	400	— —	—	—	—	—	++++	++	Ketosis, metritis. Penicillin. No improvement with dextrose	Imp.
42	W. B.	7001997	600	— —	—	—	—	—	++++	+	Ketosis, metritis. Penicillin. No improvement on previous dextrose	Rec.
43	W. B.	57965U5	600	— —	—	—	—	—	++++	++	Ketosis. No improvement on previous dextrose injection	Rec.
44	S. J.	?	200	— —	—	—	—	—	—	—	Ketosis, milk fever, retained placenta. Calcium	Imp.
45	O. S.	?	400	— —	—	—	—	—	—	—	Ketosis. Little improvement on previous dextrose administration	Imp.

[1]Rec. = Recovery; [2]Imp. = improvement; [3]Resp. = response. *A.V.U. = Armour veterinary unit.

Administration of adrenomone stimulates the adrenal cortex to produce and secrete the entire spectrum of cortical hormones at an increased rate. With the stress removed from the pituitary, it is not unusual to find some animals exceeding their milking records established before onset of the ketosis. Resumption of normal carbohydrate metabolism within three days following institution of adrenomone therapy removes the demand on body protein and fat stores as sources of carbohydrates. Blood sugar and ketone values return to normal limits, and acetone disappears from the urine.

Summary

Forty-five cows suffering from primary and secondary ketosis were treated with adrenomone. Biochemical assays of blood sugar, and blood and urine ketones were made prior to and during therapy. The treatment produced alleviation of symptoms of ketosis, with a return to normal in milk production, blood sugar and ketone values, and gain in body weight.

It is interesting to note that only eight animals in this series involving both simple (primary) and complex (secondary) ketosis required adrenomone treatment past the third day before observing either an improvement warranting no further therapy, or a recovery was affected.

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(*Continued on page 88*)

The Prophylactic Value Of Nitrofurazone In *Salmonella choleraesuis* Enteritis Of Pigs: A Field Experiment

NILS LANNEK,* D.V.M., Ph.D., and STEN BRAG,* D.V.M.,
Stockholm, Sweden

SALMONELLA choleraesuis enteritis and septicemia are serious problems in Sweden. Hog raisers who buy pigs at the age of eight to ten weeks, for meat production exclusively, are especially affected. The urgent need for a successful treatment for this disease induced us to undertake this study of nitrofurazone.

We were especially interested in the extensive experiments conducted by Guthrie[3] who found nitrofurazone effective against *Salmonella choleraesuis* enteritis in pigs when fed in doses of 0.5 to 0.6 gm. daily for two or three days, beginning one or two days after the administration of a broth culture of this bacterium.

Nitrofurazone (5-nitro-2-furaldehyde semicarbazone) was reported in 1944 by Dodd and Stillman[2] to be bacteriostatic and bactericidal against both Gram-positive and Gram-negative bacteria. It was later shown[1] to prevent death in mice infected with *Salmonella schottmülleri* and *Salmonella aertrycke*. Nitrofurazone acts in a manner quite comparable to that of the antibiotics in producing a specific disruption of enzymatic metabolism of the bacterial cell.

A survey of the literature concerning other therapeutic agents tried on this disease is given elsewhere[5] and will not be repeated here. With regard to prophylaxis, it has been reported[5] that vaccination is of no considerable value.

According to our experience it is doubtful whether vaccination with either a standard vaccine or vaccine prepared from the specific herd confers any noteworthy protection. Such vaccines were applied either only once when the pigs were bought, or two or even three times at ten-day intervals. Disinfecting procedures should be thorough and include slaughter of the whole herd to eradicate the infection.

Field Outbreak of Salmonellosis

A herd comprising about 200 pigs had been free of this disease since it was started 12 years ago. In March 1953, *Salmonella choleraesuis* enteritis appeared. Symptoms were typical. Initially, the pigs lost interest in eating and lay quietly in a corner of the pen or walked about unsteadily. After a day or two profuse diarrhea was observed. Cyanosis appeared on the ears, back, tail, and legs. Most of the herd died within a week but some recovered. Convalescent animals improved slowly. At necropsy, pigs which died or were killed revealed diphtheritic colitis and typhlitis, acute gastritis and microscopic necrotic areas in the liver. *Salmonella choleraesuis* was cultured from the viscera.

Materials and Methods

The diet throughout the experiment consisted of garbage and mixed oat, corn, and wheat meal. Animals were fed twice daily.

Nitrofurazone was used either in pure form or in a premix containing nine parts of calcium phosphate (bone ash). The drug was thoroughly mixed in a small quantity of meal and given shortly before the main feeding. The daily dose was divided in two equal parts, one in the morning and one in the evening meal. The prophylactic doses used are shown in fig. 1, ranging from 0.1 to 0.5 gm. per pig per day.

The groups consisted in each case of ten pigs. Each treated group had one control group which had been bought at the same time. When an experiment started with a treated group and its control group, all animals in these groups were obviously healthy. These two groups were kept in pens situated near to each other and given as similar feed and handling as possible except for the treatment with nitrofurazone. All cases of the disease appeared spontaneously; *i.e.*, no pig was purposely infected.

In all, 40 animals were given prophylactic treatment with nitrofurazone and 40 untreated animals were kept as controls.

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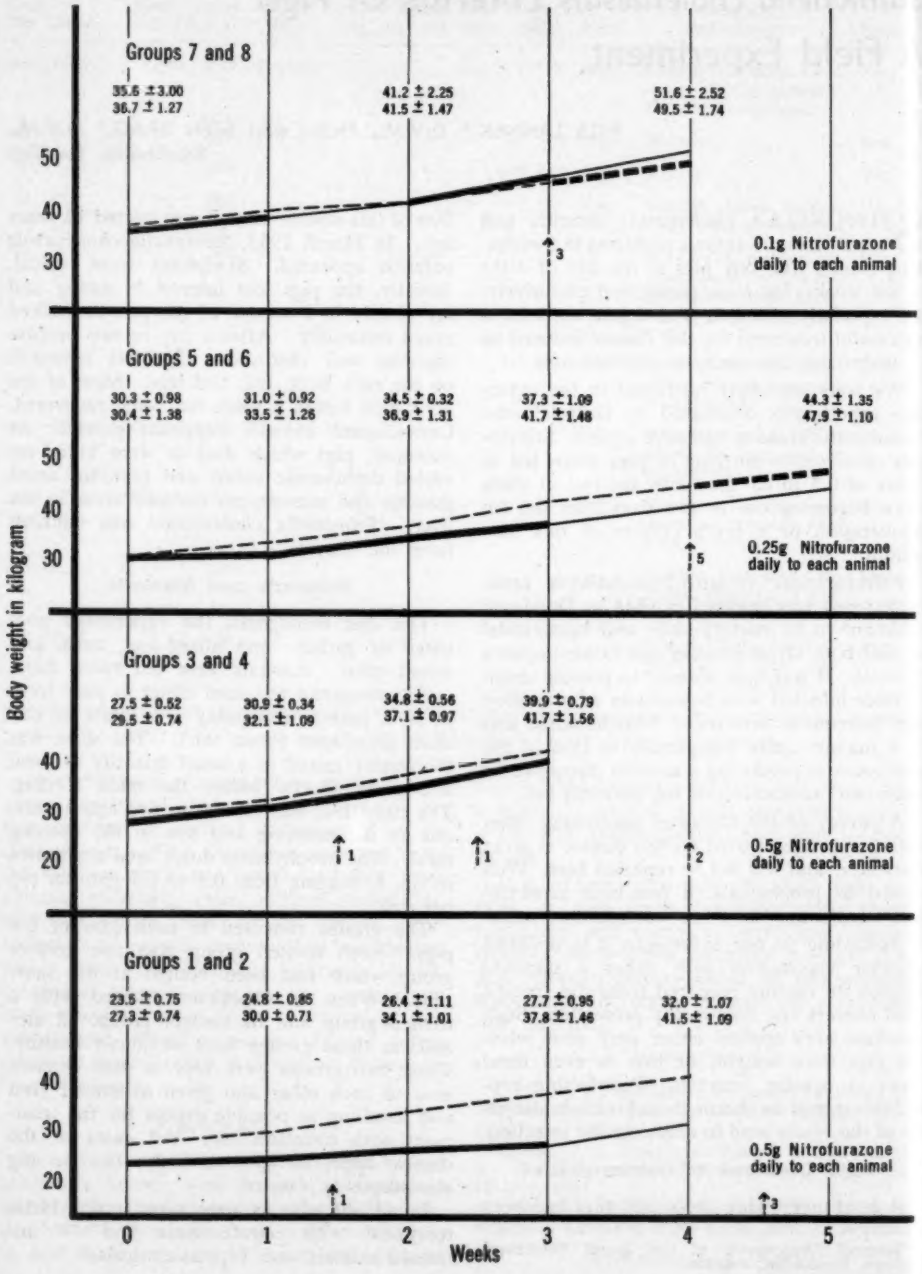
When a pig in the untreated control group showed distinct symptoms of the disease, it was removed from its group and placed in a special pen. There it was treated with the curative level of 0.5 gm. nitrofurazone daily for two or three days, and thereafter with 0.1 gm. daily, so that the whole treatment lasted one week. It was no longer included

Fig. 1



in the calculations for the group. However, when several animals in an untreated group became ill, the entire group was treated with this curative level.

Groups 1-2 and 5-6 had only spent one or two days in the herd before the experiment with them was started. The groups 3-4, on the contrary, had been in the herd for three weeks and the groups 7-8 for two weeks before they were used.

Results

The essential results of the prophylactic treatment are shown in fig. 1 and will only be mentioned in the text. So far, none of the pigs which received prophylactic treatment has shown any symptoms of the disease. One died but showed at necropsy chronic nephritis and uremia.

On the other hand, 16 of 40 nontreated pigs (40%) fell ill with symptoms of Salmonella enteritis. As mentioned previously, each pig showing distinct symptoms of this disease was brought to an isolation pen where it was treated with the curative doses of nitrofurazone as recommended by Guthrie. None of these died. Half recovered completely within a week or so. The other half of them improved only slowly and, though the diarrhea stopped after a couple of days of treatment, the appetite was poor and weight gains were slow for several weeks.

The weight diagram shows that some levels of nitrofurazone in all probability had a retarding effect on the growth rate of groups 1 and 5. It was also evident during the experiment that these groups did not have as good an appetite as did the control groups. As soon as the preventive treatment stopped, appetites became normal and the weight curves ran parallel with those of the control groups. However, groups 3 and 7 had just as good weight gains as their controls, even when nitrofurazone was given. This will be discussed later.

Discussion

It is evident from fig. 1 that the prophylactic treatment with nitrofurazone gave excellent results; none of the treated animals, but 40% of the nontreated ones, fell ill with Salmonella enteritis. This is true even for the lowest dosage level of 0.1 gm. daily for two weeks, with one week without treatment between.

Because it is a regular occurrence in our area that the disease does not break out until the animals have spent at least two weeks in the infected herd, groups 3 and 7 were not treated before three and two weeks, respectively, after they had arrived. This delay did not give any untoward effects. On the contrary this may have contributed to the fact that these animals showed quite as good appetites and growth rates as the control animals. Nitrofurazone has a slightly bitter taste and it is possible that the relatively large dose as in group 1 should decrease the appetite more than a small dose as in group 7. But it is also possible that animals, after having been fed strongly flavored garbage for some time, will accept the nitrofurazone better than animals which have until then received only food with mild taste. In the breeding herd they get mainly milk and meal. Thus, even 0.5 gm. of nitrofurazone daily did not give any adverse effect on the appetite and growth rate in group 3, as did 0.25 gm. in group 5. Group 3 had been fed garbage during three weeks before the nitrofurazone treatment was started, while group 5 was purchased only one day previously.

Apart from the temporary retarding effect on food ingestion under the conditions discussed above, no toxic phenomena were observed. According to Krantz & Evans[4] and Dodd,[1] the peroral $l.d._{50}$ in mice and rats is between 500 and 600 mg. of nitrofurazone per kg. The toxic effects were mainly symptoms arising from the central nervous system.

Fig. 1. The lines show the mean body weight of the respective groups. The start of an experiment is indicated on the abscissa by week 0. The pigs were weighed at the beginning of the experiment and thereafter at intervals of one or two weeks. Each continuous line represents a prophylactically treated group (odd group number), and the daily dose used in each experiment is given in the lower right corner of the graph. Each broken line represents a control group not receiving prophylaxis (even group number). A thick line, whether continuous or broken, indicates that the whole group was receiving two doses of nitrofurazone daily. Thus, for example, group 7 was treated prophylactically during the first and third weeks, while group 8 was treated with a curative level during the fourth week. As mentioned earlier, an entire control group was treated only after several animals in it had fallen ill and the group was considered to be seriously threatened. The treatment then consisted of the curative dose of 0.5 gm. nitrofurazone for two days and 0.1 gm. nitrofurazone during the rest of the week to each animal daily.
The small vertical arrows show when pigs belonging to the control groups without prophylaxis fell ill. The figure by the arrow shows the number of animals falling ill on that occasion. Thus, for example, three animals in group 8 fell ill at the end of the third week.
The mean weights in kilograms at the beginning of each week are given with their mean errors, above the curves. The weights of the treated groups are placed above the weights of the control groups.

Krantz & Evans found that 80% of normal men tolerate 3 or 4 gm. daily, while 20% become nauseated on this dose. Guthrie[3] did not observe any adverse symptoms when 0.15 to 0.2 gm. of nitrofurazone per kg. was given as one dose to normal pigs but 0.3 gm. or more per kg. produced inappetence, incoordination and weakness of the legs, and death. One 60-lb. pig which received a total of 24.5 gm. in doses divided over eight days showed inappetence and incoordination but recovered completely two days after the administration was stopped.

With regard to this low toxicity of nitrofurazone, it is quite improbable that the low prophylactic level used successfully in our experiment (0.1 gm. per animal daily for two weeks with one week without treatment) will produce any significant deleterious effects.

Summary

It was the aim of this field experiment to ascertain whether nitrofurazone is useful in prophylaxis of *Salmonella choleraesuis* enteritis in swine. The experiment was performed in an infected herd.

Daily doses of 0.1 gm. to 0.5 gm. of nitrofurazone per animal given over a period of two or three weeks, resulted in complete protection. None of the 40 treated animals, but 16 (40%) of the 40 nontreated control animals, fell ill with symptoms of Salmonella enteritis.

Nitrofurazone may decrease the appetite and thereby reduce the food intake and retard the growth rate. This was not observed when the pigs had spent two weeks in the infected herd before the treatment was started. It is suggested that the pigs may then tolerate the nitrofurazone taste because they have been eating the strongly flavored garbage for some time.

Acknowledgment

We are grateful to Pharmacia, Uppsala, Sweden, for supplying the nitrofurazone used in this experiment, and to Mr. Hallerfeldt, who permitted us to perform the experiment in his herd.

When this paper was written (July 1953) 12 weeks had elapsed since the experiment was started with the groups 1-2 and 3-4. The corresponding figures for the groups 5-6 are nine weeks and for groups 7-8, seven weeks.

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The Removal of Whipworms by N-butyl Chloride

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Whipworms have been considered one of the most difficult of the helminths to dislodge and eliminate from the digestive tracts of dogs, principally because of their habit of being able to live in the cecum. Whipworms in the intestines are removed by either tetrachlorethylene or n-butyl chloride, but those protected in the cecum are by-passed by anthelmintics.

In almost all of the instructions for the use of either tetrachlorethylene or n-butyl chloride, a dose of a laxative is recommended. One manufacturer includes phenolphthalein with the n-butyl chloride in his capsules.

It was our thought, after reviewing some results obtained in a study by the senior author in the administration of n-butyl chloride to puppies, that if the drug was so lacking in toxicity as it appeared, perhaps it would gain entrance into the cecum, when no physic was administered to flush it past the iliocecal valve into the colon.

We had already seen that without a physic following, a dose of four times that found necessary to eliminate ascarids and hookworms, but given in split doses, was without apparent toxicity to puppies[1]. As stated in the previous paper, we found it necessary to precede the usual whipworm dosage of n-butyl chloride with an antiemetic in order to assure that this increased dosage is retained. Dosing results had been fairly satisfactory since using the divided dose system.

We also learned from consulting many before-and-after reports of fecal examinations[2] that we were having far better results when no physic was given following its administration, even though this was not in accord with previous recommendations. It was decided, (based on this fact and coupled with our knowledge of the lack of toxicity and nausea

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Clinical Laboratory Diagnostic Procedures In Small Animal Practice†

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A questionnaire listing 14 possible subjects for discussion was sent to a group of Florida practioners. The replies indicated that most were interested in the subject here discussed, clearly indicating that the field of veterinary practice is undergoing some rapid transitions. Veterinarians are now thinking in much more highly technical, biochemical, and physiological terms in relation to problems of daily practice.

In 1930, we were unable to diagnose many of the diseases of dogs and cats which were brought to our attention. In addition to microscopic examinations for parasite ova, it was decided at that time to employ additional clinical laboratory tests which included Schilling blood counts and urine analyses. Experience soon demonstrated that at least five factors were important:

1. What tests could be performed on a given patient to produce the desired information?

2. What interpretation could be made of the results on that particular patient?

3. What could be done, therapeutically, to correct the apparent pathological findings?

4. What change in diet was indicated to produce a therapeutic response?

5. What recheck tests should be performed to follow effectively the animal's response or lack of response?

Twenty years experience has indicated that the determinations which are tabulated herein were those most useful in making diagnoses and selecting therapeutic procedures in the practice of small animal medicine.

CLINICAL LABORATORY DIAGNOSTIC PROCEDURES FOUND PRACTICAL and COMMONLY EMPLOYED

Section A

Blood
- Hemoglobin
- Erythrocyte total
- Leukocyte total
- Differential white count
- Dirofilaria
- Blood sugar
- Blood calcium
- Blood phosphorous
- Blood urea
- Blood cultures

Urine
- Reaction
- Albumin
- Sugar
- Blood
- Indican
- Sediment
- Specific gravity
- Leptospirosis
- Uric acid

Feces
- Respiratory parasites
- Alimentary tract parasites
- Spurious parasites
- Foreign material such as starch granules, grain mites, undigested fat

Skin
- Mange
- Fungus
- Sections

Tumors
- Biopsies before surgery or *x*-ray

†Presented at the Florida State Veterinary Medical Association annual meeting, Daytona Beach, Oct. 18-20, 1953.

SUGGESTED DETERMINATIONS

Section B

Urine

Urine ash
Quantitative uric acid
(Published as part of this report)

Feces

Rapid method for fat determination
Examination of gross sample
Color, consistency, volume in relation to diet, mineral matter, undigested cellulose, blood.

Discussion

The above has been divided into sections A and B. Section A is concerned with those determinations which were employed routinely and found useful and applicable. Section B contains suggested determinations which would appear to have usefulness in view of recent developments in the field of small animal practice.

Comments on Section A

Certain determinations may be thought of as companion tests. A total erythrocyte count, which includes a hemoglobin determination, is more valuable than either alone. A total leukocyte count, together with a differential white cell count, provides valuable information not available from a single determination. Blood sugar and urine sugar combinations are most desirable. Blood urea is more useful and can be interpreted more accurately when considered with the specific gravity of the urine. This combination has merit as one kidney function test. The reaction of the urine, whether acid or alkaline, often can aid in determining the need for urine culture. Albumin, less important in the dog and cat than in man, is of distinct aid in appraising kidney function. The amount of urine uric acid eliminated by the dog is one method for appraising liver function. The laboratory method is included as a part of this manuscript.

Comments on Section B

The percentage of mineral ash in the urine of dogs and cats has become important since many of these animals are being fed animal foods which contain large excesses of mineral matter, some of which is absorbed and appears in the urine as waste ash. Such a test, therefore, can serve a useful diagnostic purpose and be a guide in outlining proper therapy.

Microscopic examination of feces for parasite ova is a common practice; however, much additional information can be obtained from a more thorough examination of gross specimens. Such examinations should include color, consistency, volume in relation to the diet consumed, presence of large amounts of mineral matter, undigested cellulose, fiber, blood cells, *et cetera.*

A rapid method for the estimation of fecal fat in the dog would also be useful. Some research is being carried out to develop this essential test.

Liver Function in the Dog

There are a number of diseases of dogs which may be responsible for producing chronic liver injuries. Symptoms often develop months or years later, for example, ascites. A single test which will appraise the various liver malfunctions will in all probability never be devised. However, for a number of years we have employed the quantitative urine uric acid test, which was suggested to us by Doctor Bollman of the Mayo Clinic, and have found it very useful in many instances. The laboratory technic is rather involved, but for those who are interested, the procedure as modified by Dr. J. B. Allison of Rutgers University has been found accurate and clinically satisfactory.

Determination of Uric Acid in Urine (Folin modified by Allison)

Reagents and Preparation of Reagents.

Chloride — acetate solution
5% Silver nitrate solution
50% Urea solution
12% Sodium cyanide solution (poison)
Standard uric acid solution
Uric acid reagent

Chloride-Acetate Solution. Dissolve 1 gm. sodium chloride, C.P., and 2 gm. of crystallized sodium acetate, C.P., in water, using about 50 ml. Add 1 ml. of glacial acetic acid and dilute to 100 ml.

5% Silver Nitrate. Dissolve 5 gm. silver nitrate, C.P., in water to make 100 ml.

Urea-Cyanide Solution. In a liter beaker place 50 gm. of sodium cyanide, and add 700 ml. of water. Stir until solution is complete. Add 300 gm. of urea, C.P., and shake moderately for five minutes. Add 2 gm. of calcium oxide, and let stand for one hour. Filter off the precipitate and add to the filtrate 0.5 gm. of lithium oxalate. Let the solution stand overnight and filter the next day. Keep well stoppered to prevent absorption of carbon dioxide.

(*Continued on page 84*)

A New General Anesthetic For Small Animals†

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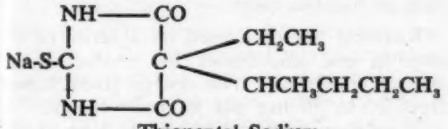
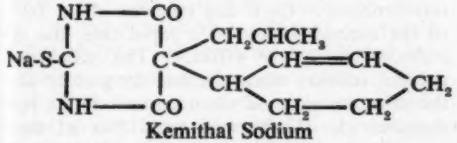
GENERAL anesthesia may be produced by two principle methods; that is, use of volatile anesthetics such as ether, chloroform or trichlorethylene, or use of nonvolatile anesthetics which would include barbiturates. Several of the latter have been used effectively in small animal practice for several years and consideration of another would not be advantageous unless the new drug possessed special properties or superiorities.

A new intravenous barbiturate, kemithal,® was developed in the laboratories of Imperial Chemical Industries, Limited. Carrington *et al.*,[1] reported on the pharmacology of this new agent and compared it with thiopental sodium. Krantz,[2] reported in 1951 that thiopental was one of the most widely used nonvolatile or fixed anesthetics. The following formulas may be presented to indicate the differences in thiopental and kemithal:



Thiopental Sodium



Kemithal Sodium

Kemithal is the sodium salt of 5-$\Delta^{1':3'}$-cyclohexenyl-5-allyl thiobarbituric acid. It is a pale yellow powder, readily soluble in water; the 10% solution has a pH of about 10.6. Solutions are stable for seven days when stored at ordinary refrigerator temperatures. Equiactive doses of kemithal and thiopental produce similar duration of action and onset of anesthesia. Daily injections of kemithal in rabbits for a period of 30 days did not produce any detectable histological changes. It was found to depress the respiratory volume less than thiopental; anesthesia can be maintained with less respiratory risk. Kemithal is almost completely detoxified by body tissues. Another advantage in its use is the observed decrease in liklihood of laryngeal spasm[3] which has been observed in small animals under thiopental anesthesia.

A number of veterinarians in Europe have described the successful use of this product for anesthesia of small animals. For example, Beijers,[4] used kemithal in a large series of animals intravenously in 10% solution. Doses given ranged from 6 to 12 cc. per animal and the duration of anesthesia averaged approximately two hours. He also developed a satisfactory technic for administering this agent to cats. Beijers gave 2 to 4 cc. of 10% solution intraperitoneally. He reported that anesthesia was produced in four to five minutes and was of "good quality", the cats lying quietly in full anesthesia. Voute and Hannema,[5] also proposed the intraperitoneal route as a practical method of administering kemithal in cats. Compared to thiopental it was found superior because of its less depressant action on the respiratory center.

Seneviratne and Pillai[6] used kemithal as the intravenous anesthetic of choice in over 130 cases for major operations on dogs. The following advantages of this new anesthetic over other barbiturates were cited: (1) No period of excitement prior to anesthesia, (2) anesthesia complete in the course of one to three minutes which was the duration of the injection, (3) anesthetic given much more rapidly than sodium pentobarbital, (4) degree and period of anesthesia varied from 15 to 45 minutes depending on the dosage, (5) recovery after anesthesia generally much more rapid than after other barbiturates, (6) little excitement during or after recovery. The dosage schedule recommended by these veterinarians was as follows: Below 10 lb. bodyweight—30 mg. per lb.; from 10 to 30 lb.—20-30 mg. per lb.; from 30-50 lb.—15-20 mg. per lb.; over 50 lb. —15 mg. per lb. Anesthesia was continued up to one and one-half hours by injection of one-third the original dosage as needed at subsequent intervals. The following changes in the animal were observed during anesthesia: During deep anesthesia, the pupil was partly contracted and pulse accelerated, while res-

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*Ft. Dodge Laboratories.
®Ft. Dodge Laboratories, Ft. Dodge, Iowa.

pirations were deep and slow; there was no salivation, defecation, or urination, and visible mucus membranes were congested. These workers concluded that kemithal was a safe and efficient intravenous anesthetic for dogs, to be used in operations lasting up to one and one-half hours. It was demonstrated to be less toxic than other barbiturates and there was no preanesthetic excitation, while the period of preanesthetic narcosis was short. The toxic dose appeared to be more than one and one-half times that required to produce anesthesia of short duration.

Harrow[7] reports that two veterinary practitioners in Scotland have each anesthetized over 1,000 dogs and cats with kemithal and prefer it for all surgery. A dosage of 50-100 mg. per kg. body weight (20-45 mg. per lb.) produced complete anesthesia. The smaller dosage gave surgical anesthesia for 10 to 15 minutes, and the larger gave anesthesia lasting up to three hours. After anesthesia, animals recovered quickly and period of ataxia after the first signs of recovery appeared was shorter than observed after the use of other barbiturates. Compared with thiopental, it was considerably less depressant to the respiratory centers. A dose of 2.5 mg. per kg. of thiopental produced a depression of respiratory volume equivalent to that produced by 8 mg. per kg. of kemithal.

Stockton,[8] reported successful use of barbiturates by intrapleural or intrathoracic administration. Animals were weighed and the estimated dosage drawn into a sterile glass syringe fitted with a 22 gauge, one-inch needle. An injection area on either the right or left side between the seventh and eighth, or eighth and ninth, ribs was prepared for injection. The needle was introduced straight in to a depth of one-fourth inch. Negative pressure was applied until a bubble appeared in the syringe. The solution was then injected intrapleurally. Preanesthesia excitement was reduced and frequently was entirely absent, with light surgical anesthesia being induced by the time the animal was returned to its cage.

Recently, Doctor Barnes[9] has reported on the successful use of kemithal intrapleurally for cats. The recommended dosage was 30 to 40 mg. per lb. body weight. He has reported the development of good surgical anesthesia in four to five minutes with an average duration of two to three hours. There have been no complications. A dosage of 40-50 mg. per lb. body weight given intrapleurally to five cats in our laboratories produced prompt anesthesia in five minutes. Respiration was regular and deep, and anesthesia continued for an average of 50 minutes.

Preanesthetic Preparation

The thiobarbiturates are characterized by causing some increase in salivary flow. This stimulatory action has been reported by numerous veterinarians using thiopental sodium. Some have reported a sialogogue action of kemithal on dogs. To inhibit this action of either of these two barbiturates, atropine sulfate frequently is administered subcutaneously, in dosages of 1/300 to 1/150 gr., 15 minutes prior to anesthesia. This not only depresses the secretion of saliva, but also depresses reflexes mediated through the vagus, counterbalances the respiratory depression of morphine, if it has been used, and is reported to produce some psychic sedation.

Certain practitioners have inquired about the advisability of using morphine as a preanesthetic sedative prior to the administration of kemithal. Such usage is recommended when the following are definitely indicated: Reduction of reflex irritability and metabolism, and as an analgesia. Morphine is recognized as being a poor hypnotic and because of its depressant respiratory action should be used only in selected cases.

Kemithal has been used on a series of 60 dogs in our laboratories for production of general anesthesia. The dosage given varied from 20 to 50 mg. per lb. body weight. A 10% solution, prepared by adding 1 gm. kemithal in 10 cc. of water or saline was injected intravenously. Each dog received about 60% of the estimated dose at a rapid rate, the remainder slowly "to effect." The pedal and corneal reflexes were checked frequently and the injection stopped when corneal reflex had disappeared. There was rapid loss of consciousness with little or no excitement and pupils were contracted when the stage of surgical anesthesia was reached. This, in itself, was interpreted as indicating continued activity of the sympathetic nervous system and good surgical condition of the animal. Records were taken of the pulse and respiratory rates prior to and at five-minute intervals during the period of anesthesia.

Another uniform clinical observation was excellent relaxation of the abdominal wall, a quality of particular advantage in an anesthetic for abdominal surgery. In the use of some other available barbiturates, poor muscular relaxation is noted and intestines tend to push up and out of the abdominal incision. Some of these also cause irritation, necrosis,

and sloughing if injected subcutaneously, or if inadvertently injected outside the vein during intravenous administration. Kemithal solutions, being less irritant, cause little or no tissue reaction should perivascular leakage occur. In those dogs receiving a dosage of 50 mg. per lb., bradycardia was evident during the first five minutes of anesthesia. When the amount was reduced to that level producing good surgical anesthesia, falling within the range of 20-40 mg. per lb., there was little evidence of cardiac or respiratory disturbances.

Because of reports of salivary stimulation by the other thiobarbiturates, possibility of combining atropine sulfate therapy with kemithal was investigated. Atropine (1/300 to 1/150 gr.) was given 15 minutes prior to injection of the anesthetic agent. There was little or no alteration in the expected anesthetic effectiveness in a series of 12 dogs. In our own experience, we have seen no dog for which the inclusion of atropine sulfate would be indicated.

Use of Combined Curare

Because of the inadequacies of older barbiturates, surgeons in medicine and veterinary medicine have combined curariform agents in order to reduce the total amount of anesthetic needed for surgical procedures. Curare, when originally introduced into medicine, was a mixture of the active ingredients of a number of poisonous plants and was described as a powerful poison, the main action of which was to paralyze skeletal muscles[10]. In 1935, a highly active principle was isolated from curare which was named *d*-tubocurarine chloride. At the present time, solutions or crystals of this material are available for clinical use.

Curare produces muscular relaxation by blocking the transfer of the effector impulses to the skeletal muscle at the neuromuscular junction. The drug probably raises the threshold of the muscle fiber to the action of acetylcholine. When curare is combined with the older barbiturates, it assists in retraction of the intestines within the abdominal cavity in abdominal surgery by causing relaxation of the skeletal musculature rather than by increasing the tonus of the intestines[11]. The injection of curare intravenously is followed within two minutes by muscular relaxation. Its action is selective since it first depresses the muscles of the head and neck region, then the muscles of the body and legs, and last those of respiration, the diaphragm being the last muscle to be paralyzed. Recovery is in reverse order[12].

Curare has been used in combination with thiopental sodium. Such a combination is advantageous, since this barbiturate is a parasympathomimetic drug and enhances the laryngeal and bronchial reflexes with predisposition to laryngospasm in abdominal surgery.

Doctor Knight[13] of the University of Minnesota Hospitals, developed a method of giving thiopental and curare in a fixed ratio in conjunction with nitrous oxide and oxygen by inhalation. Baird[14] reported on the compatibility of 2.5% thiopental sodium and *d*-tubocurare. The solution was prepared to contain five units of *d*-tubocurarine per cc. of 2.5% thiopental. This ratio proved satisfactory in the majority of cases. Since thiopental and *d*-tubocurare are both known to cause excessive salivation, it is wise to premedicate animals receiving these agents with morphine and atropine, or atropine alone. The ratio of morphine and atropine is generally given as 25:1. Cullen[15] reported that curare in unmedicated dogs caused profuse salivation with marked respiratory difficulty and muscular twitching.

With this report in mind, we believed it wise to investigate the possibility of using curare in combination with kemithal in animal surgery. In reviewing the literature concerning further information available on the use of curare, we found that Pelikman[16] reported that a dosage of 0.3 mg. of *d*-tubocurare given intravenously caused "head drop" in cats and rabbits within two to three minutes. One half of this dosage repeated in 45 minutes again caused "head drop" and with minor exceptions the effects of the "fill-up" were the same as the initial one. Thus, the presence of *d*-tubocurare in the body, after apparent recovery from the paralytic effects, was demonstrated in experimental animals and human patients.

Brinker[17] suggested administration of surital[®1] sodium at a dosage rate of ⅛ gr. per lb. which produced surgical anesthesia lasting 15 minutes. Surital sodim alone markedly stimulated the flow of saliva, but when combined with *d*-tubocurare, salivary stimulation was decreased. A series of animals received 1 cc. per 5 lb. bodyweight of a mixture containing 40 mg. of surital and ⅜ unit of curare which produced satisfactory surgical anesthesia.

Similarly, Pickett[18] reported that a combination of nembutal,[®2] ¾ gr. per 5 lb., and curare, ⅜ unit per lb. in dogs gave complete muscular relaxation up to one hour. He stressed the fact that 95% excellent results and 5% mortality rates were not acceptable to owners of dogs or cats. The anesthesia should be safe and effective with no mortality

[®1]Parke, Davis Company, Detroit, Mich.
[®2]Abbott Laboratories, North Chicago, Ill.

from the combinations of drugs used.

In a group of nine dogs in our laboratories, good surgical anesthesia was produced by combining *d*-tubocurare, ⅜ unit per lb., with 20 mg. of kemithal per lb. body weight. These dogs received 1/300 gr. of atropine 15 minutes prior to administration of combined kemithal and *d*-tubocurare.

Gray[19] reported on the use of kemithal in 30 cases of cesarean section when combined with *d*-tubocurare; he found a virtual absence of respiratory depression in infants born to mothers anesthetized with this combination. Corbett and Thomas[20] used Gray's technic in a series of 100 cases and concluded that uterine contractility, minimal blood loss, absence of toxicity, operative convenience, and safety to the infant are reasons for further trials with this product.

Numerous clinical reports on the use of kemithal alone for cesarean sections in bitches have been submitted in which the veterinarian reported using the minimal effective dosage with a resulting high survival rate of dams and puppies.

Summary

The new nonvolatile anesthetic, kemithal, satisfies more of the criteria for a good general anesthetic for use by veterinarians than other barbiturates introduced thus far. Practitioners are advised to use proper caution at the outset when beginning to work with any new anesthetic. Because of the greater safety of kemithal, it is believed that this new product is another excellent "tool" in the armamentarium of the practitioner specializing in small animal medicine.

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Diagnostic Procedures

(*Continued from page 80*)

Stock Uric Acid Solution. On a watch glass weigh out 0.2 gm. of pure recrystallized uric acid. Transfer this through a dry funnel to a dry 200 ml. volumetric flask. Prepare a solution of 0.15 gm. of lithium carbonate in 30 ml. of water, warm to 60 C. and pour over the watch glass and through the funnel into the flask, previously warmed under the hot water tap. After shaking for five minutes, the acid should be dissolved. Cool under running water and add 4 ml. of neutral formalin and 70 ml. of water. Now add gradually 10 ml. of N/2 sulfuric acid, with continuous shaking. Dilute to 200 ml. and mix well, store in well stoppered bottle away from light.

Uric Acid Reagent. In 500 ml. flask put 100 gm. of molybdate-free sodium tungstate and add 32-33 ml. of 85% phosphoric acid which has been mixed with 150 ml. of water. Shake thoroughly, add a few glass beads and boil gently with a suitable condenser for one hour. Decolorize with a little bromine water, boil off the surplus bromine, cool, and dilute to 500 ml.

Standard Uric Acid Solution. Dilute 1 ml. stock uric acid solution to 250 ml. of water.

Method of Procedure

1. Dilute 5 cc. of the 24-hour urine sample, plus 10 cc. of chloride-acetate solution, to 100 cc. with distilled water.

2. Place 5 cc. of the diluted urine in a centrifuge tube (equivalent to 0.25 cc. undiluted urine).

3. Add 3 cc. of the 5% silver nitrate solution. Mix by swirling tube *not* by stirring. Centrifuge five minutes at 1,760 r.p.m. or higher.

4. Decant supernatant fluid. Drain tube 30 — 60 minutes.

5. Add 2 cc. of the 12% sodium cyanide solution from burette to tube. *Do not pipette.* Measure this *very* carefully and accurately. Mix well to dissolve, using a glass rod which is left in the tube and solution.

6. Add 2 cc. of the 50% urea solution by *accurate* pipetting. Mix with the glass rod already in the tube which is not removed.

(*Continued on page 86*)

Nutritive Requirements Of The Cat. II.*

MARK L. MORRIS, B. S., D.V.M., Topeka, Kansas

The November 1953 issue of this publication carried a report entitled, Nutritive Requirements of the Cat: A Preliminary Report, which was presented at the annual meeting of the American Animal Hospital Association, Minneapolis, Minn., May 6-9, 1953. In the discussion paragraph it was stated that, "We should know definitely whether it is possible to feed standardized cats on a canned processed diet that has been sterilized according to known specifications." In the same paragraph is the statement: "The quality or biological availability of ash present is important, as well as the quantity present."

For consideration at this time are the feasibility of and physiological reaction to suddenly changing a colony of standardized cats from a freshly prepared, raw meat-like diet, designated "RB2", to a carefully balanced but sterilized canned product designated "c/d".

TABLE I. Change in Food Intake on Switching from a Fresh Meat to a Canned Low Ash Diet

Date	Diet	Food offered ea. cat (gm.)	No. cats refusing part of food	Av. amt. refused (gm.)	Av. food consumption for colony (gm.)
Aug.					
5	RB2	135	0	—	135
6	RB2	135	0	—	135
7	RB2	135	0	—	135
8	RB2	135	0	—	135
9	RB2	135	0	—	135
10	RB2	135	2	93	121
11	RB2	135	2	88	122
12	c/d	148	3	100	112
13	c/d	148	3	93	127
14	c/d	148	1	60	143
15	c/d	148	3	92	127
16	c/d	148	1	70	143
17	c/d	148	0	—	148
18	c/d	148	1	70	143
19	c/d	148	0	—	148
20	c/d	148	4	73	126
21	c/d	148	1	100	140
22	c/d	148	1	40	145
23	c/d	148	2	60	139

Discussion of Table I

In the column entitled, Food offered each cat (gm.), it will be noted that the average amount of RB2 fed was 135 gm., the average amount of c/d fed was 148 gm. The amount of moisture present in the two rations was not constant, thus, in order to maintain a reasonably accurate energy intake, it was decided to increase all cats to an average daily intake of 148 gm., regardless of body weight. The column entitled, No. cats refusing part of food, refers to the total number of the 13 standardized animals fed which returned even a small portion of a 24-hour feeding.

It will be noted that on August 11, all cats received the regular 135 gm. feeding of RB2 (raw, fresh food). On August 12, all cats received 148 gm. of canned sterilized food. On that day, three of 13 cats refused a portion of the food amounting to 100 gm. On the same date, the average consumption of food for the colony was 112 gm. out of 148 gm. fed, compared with 122 gm. average intake on the previous day, on which date there were 88 gm. refusals on the regular RB2 feeding. Subsequent experience with this colony has indicated that cats weighing from 3 to 4 kg. will not consume regularly a total of 148 gm. of food. This proved to be more digestible energy than was required by animals of that weight. This point should be considered in appraising the data above.

With this colony consisting of 13 male castrate, well nourished animals, it was possible to transfer directly from the fresh, raw RB2 diet to the canned sterilized product designated c/d without significant disturbances in appetite, food consumption, or bowel movement.

TABLE II. Food Consumption and Ash Excretion of Cats on a Fresh Meat and Canned Low Ash Diet

	Diet	
	RB2	c/d
Average Daily Food Intake		
Gm. per kg. body weight	24.8	27.61
Calories per kg. body weight	53.8	43.1
Range in calories per kg. body weight	49.0/ 74.6	35.4/ 59.4
Average Urinary Ash Excretion		
Gm. ash consumed in each 100 gm. of food (% ash in diet)	1.8	0.82
Gm. ash excreted per 100 gm. food consumed	0.81	0.41
Ash balance—% (Excretion x 100) / Intake	45.0	50.0

Discussion of Table II

This table presents some interesting and significant information. First, it should be noted that it required 24.8 gm. of RB2 per

*Presented at the Florida State Veterinary Medical Association annual meeting, Daytona Beach, Oct. 18-20, 1953.

kg. per day, compared with 27.6 gm. per kg. on the canned product. When we examine the calories per kg., however, it will be noted that on RB2, 53.8 calories per kg. per day were required, while on the more accurately balanced canned sterilized product, 43.1 were required. Line 3, shows the range in calories consumed, with the narrower range in the case of the sterilized diet.

The information in which we are all interested, and one of the basic reasons for establishing the colony of standardized animals, is the correct basal information on the animal ash excreted by a normal cat in relation to the diet consumed. It will be noted that cats consuming the fresh RB2 ration were obtaining 1.8 gm. of ash for each 100 gm. of food consumed. Those on the low ash, canned sterilized diet were receiving but 0.82 gm. of ash per 100 gm. of food consumed. The next, and the most significant point, is how much ash appears in the urine as waste? On RB2, the cats eliminated 0.81 gm. per 100 gm. consumed; on the low ash canned food, 0.41 gm. Thus, on the canned sterilized food, the animals are eliminating just above one-half the amount of mineral waste as when fed the fresh meat type diet.

Interpretation for the Clinician

When cats are presented to veterinarians as patients exhibiting symptoms suggestive of urinary incontinence, obstruction, calculus formation, urethritis, cystitis, et cetera, it is important to know whether the disease is associated with improper excretion of mineral waste in the urine, lack of dietary vitamin A, atrophy of the urethra sometimes associated with early castration, or bacterial infections of the urinary tract. With these data to serve as guides, it should be possible to collect a representative sample of urine, dessicate it to dry ash, and thus estimate the approximate amount of nonutilizable ash excreted by the animal. This will be one step toward the more accurate differential diagnosis of urinary involvements in cats. The practitioner should keep in mind that the majority of cats today are being fed prepared and fresh rations which supply large excesses of nonutilizable mineral which will subsequently appear in the urine as ash. Some of the foods commonly consumed by cats, which are high in dietary ash, are canned whole fish products, fresh or canned horsemeat which contains quantities of green bone, dry foods such as meals, pellets, et cetera, that are fortified with meat and bone scrap. In the clinical management of urological problems in cats, the following points are important:

1. Has the dietary vitamin A intake been adequate?

2. Has the cat been consuming a diet excessive in mineral ash?

3. Is there a bacterial infection of the urinary bladder and urethra?

4. Has the cat been on a biologically unbalanced ration consisting chiefly of low quality protein, insufficient fat and inadequate amounts of vitamin B complex?

These studies which are being continued and on which subsequent reports will be published, clearly indicate the role of nutrition in the management of urinary diseases of cats.

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Diagnostic Procedures

(*Continued from page 84*)

7. Add 1 cc. of the uric acid reagent. Mix with rod. Wait 20 minutes, then add 10 cc. distilled water. Mix with rod. Compare with the standard uric acid solution in the photoelectric colorimeter.

8. Treat 1 cc. of standard uric acid solution (0.004 mgm. per cc.) but, in step no. 7, add 9 cc. distilled water instead of 10 cc.

9. Using 650 mm. filter in photoelectric colorimeter, calculate results using:

$$\frac{\text{Unknown sol.}}{\text{Standard sol.}} \times .004 \times \frac{\text{Volume of 24 hour urine sample}}{.25} = \text{Total mgm. for 24 hours}$$

The normal 20 kg. dog should excrete less than 100 mg. per 24 hours (about 5 mg. per kg.) — the Dalmatian excepted.

Clinical interpretations of laboratory findings require training, experience, skill, and the art of practice. In every instance, results must be interpreted in terms of the individual patient on which the samples were collected. This necessitates an accurate history, careful physical examination, and the possible impact of therapeutic substances given the animal prior to the collection of samples. A dietary history is exceedingly important, as is the degree of hydration. A single group of determinations may serve to misinform. It is often necessary to have results on several samples collected under different conditions before an accurate appraisal of the physiological state can be made. Clinical laboratory tests are delicate and useful instruments in the hands of the experienced who have a knowledge and respect for the values and the limitations.

EDITORIAL COMMENTS

Too Close to the Grindstone?

Wherever veterinarians meet for group discussion, it becomes apparent that all recognize the need for furthering public relations efforts. Virtually all effort heretofore has been directed toward advancement professionally. Too little effort has been devoted to creating a public awareness of the scope of veterinary science and its importance to Mr. and Mrs. America. Veterinarians have been inclined to work behind the closed door of the laboratory and clinic and to so apply themselves to the problems presented that the public might benefit through services they were trained to perform. To some it has not been considered ethical to do otherwise. These precepts are not infallible. Promotion efforts can, and should, be made by individuals or organizations representing professional groups by an open-door policy of education of what veterinary service means to human health and to the national economy.

The lack of understanding of the scope of duties the veterinarian performs, and the significance of his efforts for the national good, is appalling. Such lack exists even in high places. Too common is the attitude of the man-on-the-street that "since I own no horse, I need no veterinarian."

A challenge exists—we must devote more time to public relations work and to making our purpose and the services we can render better known to Mr. John Q. Public.

Notable work has been accomplished, and greater things are in promise, by extended programs of the American Veterinary Medical Association, Associated Serum Producers, various state associations, and some few other groups. Certain individuals also have taken advantage of opportunities to appear before lay groups, such as local Chambers of Commerce, service clubs, and similar organizations. More are encouraged to do so. No dramatics are needed. Facts speak for themselves. It usually is sufficient to point out only a few facts to elicit a surprised reaction or a remark, such as "I didn't know veterinarians were so employed, or that anything important to me or my family were of special concern to them." A few remarks about some situation or circumstance that " hits close to home", such as rabies control in the community, or better inspection of the local milk supply, will arouse the interest of even the most apathetic. The control of animal diseases which are transmissible to man, the production of healthy animals for meat and meat products, sanitation for human health in the handling of livestock products of the farm, and the treatment and care of pets and prized animals, are but a few of the subjects upon which an educational program for public information might be based.

Extension of efforts toward education of the public should be the goal of all segments, individuals, and groups in the profession. In addition, we, as individuals, can "advertise" our profession by demonstrating improved proficiency and sincere concern, at all times, in problems of a particular client or community. There are many ethical ways favorably to influence public opinion of ourselves and of our services.

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Frozen Semen Banks

Long-term preservation and long-distance shipment of bull semen, including international exchange, is now approved. Latest reports on storage gives evidence that, when semen is diluted and glycerine is added, spermatozoa resume motility after storage for one year at —79 C. Furthermore, there appears to be no loss of fertility.

Technic is too new for final appraisal. However, storing semen collected from meritorious bulls for long periods offers significant advantages. Fewer bulls will be required; it will make possible breeding out undesirable dominant or recessive inheritance factors, and speed upgrading for milk and meat production.

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Granular vaginitis is an infectious venereal disease of cattle and usually is a herd problem. It is recommended that the entire herd, including the bull, be treated if any animals are found to be affected.

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By means of intranasal intubation of the feline, up to 50 cc. ot nutrient media can be introduced into the stomach. Amounts exceeding this are apt to initiate vomition.—*C. H. Clark, D.V.M., Columbus, Ohio.*

Significant Livestock Losses Due to Careless Handling Methods

Bruising of cattle and hogs continues to occasion extensive financial losses to livestock producers. A survey indicates that of 163,444 head of cattle received at central stockyards, 10,414 were bruised to such extent as to cause a monetary loss of $5.71 per bruised animal. Of 775,332 hogs, 65,289 had bruises, with a resulting $0.95 monetary loss for each bruised hog. Properly constructed equipment to move animals to market, avoidance of indiscriminate mixing of weak and strong, large and small animals, and patience in loading and unloading will prevent much of this loss. Veterinarians can advise and aid in such a program of conservation.

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Life Span of Human Leukocytes

A recent study by Kline and Cliffton (*Jour. Applied Physiol.*, [Aug.], 5:79, 1952), in which radioactive phosphorus (P^{32}) was built into the nuclei of cells, accurately establishes that the life of human leukocytes is 13.2 days.

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Relationship of Vitamin A to the Development of Hyperkeratosis in Calves

Hyperkeratosis was produced experimentally by feeding processed feeds containing a toxic, hyperkeratosis-producing factor*.

It was observed that the factor producing this disease had a severe depressing action on vitamin A concentrations in blood plasma. There appeared to be a definite and direct hyperkeratosis factor-vitamin A relationship.

Supplementation with vitamin A (25,000 I.U. daily) throughout the feeding period resulted in greater livability of experimental animals but it did not prevent the disease or its fatal termination. If the disease was recognized early, and source of toxic material removed from the diet, supplementation of vitamin A was beneficial.

Determination of blood plasma vitamin A concentrations was helpful in diagnosis of hyperkeratosis and in following its development and regression in experimental calves.

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Ketosis

(*Continued from page 74*)

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Removal of Whipworms

(*Continued from page 78*)

when one-fourth of the whipworm dose was given every hour, after 24 hours fasting), to introduce this method into our general practice for use in dogs of all breeds, sizes, and ages, in the attempt to eliminate whipworms.

This is a procedure which clients can carry out at home with excellent results assured. When dogs are found with hookworm or roundworm infection, the usual dose is prescribed. When whipworms also are present, as revealed by fecal examinations, we give or recommend to those who wish to deworm at home, five times the dose of n-butyl chloride, but split into four doses, one given every hour, or putting it another way, we recommend the ordinary ascarid-hookworm dosage once an hour for five doses. Food is given four hours after the last capsule. No physic is administered.

Clients return a week later with a sample of stool for verification of results. The percentage of whipworms eliminated is close to 100% as gauged by ova count in centrifuged sodium nitrate ($NaNO_3$) solution floatation.

Several hundred dogs have been safely and satisfactorily dewormed of whipworms in this manner, many showing 100% elimination. The dosage also eliminates hookworms and roundworms as well. Quantities prescribed depend on body weight and are in accordance with charts prepared.[1]

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PRINCIPLES OF ANIMAL HEALTH

R. R. DYKSTRA, D.V.M.
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Tons of Moisture from Hens

One hundred hens in high production produce from five to five and one-half gallons of moisture each day. Stated otherwise, this means that during the four months, extending from November 1 to March 1—the months that high producers are more or less continuously housed, they produce over two and one-half tons of moisture. It is estimated that one-third of this moisture is in the exhaled air and two-thirds in the droppings (poultry having no urinary bladder, urates are discharged with the feces). The significance of all this is that there must be adequate ventilation, and if used, deep litter must be dry if maximum production and health are objectives during the winter months.

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Closure of the Ductus Arteriosus and Foramen Ovale

An interesting and informative dissertation, entitled Circulatory Adaptations to Birth, appears in the October 1953 number of the *Scientific Monthly*. In general, two questions are raised: (1) When and why does the ductus arteriosus close? (2) When and why does the foramen ovale close? As is well known to veterinarians, during fetal existence the ductus arteriosus is the vessel that shunts the blood, in part, between the pulmonary arterial trunk and the aorta, and the foramen ovale is the window or opening in the fetal heart between the auricles.

By means of research on lambs at the moment of full-term birth, under conditions simulating intrauterine conditions, it was determined that the blood pressure, at the beginning of the ductus arteriosus in the pulmonary artery, exceeds that in the aorta at the other end of the ductus. Almost immediately after air enters the lungs there is a "profound fall in both the pulmonary arterial and aortic blood pressures". These changes take place with the first filling of the lungs, and as a result, the ductus arteriosus is closed in a minute or two after aeration of the lungs. It is hypothesized that the ductus, being composed of elastic and muscular tissue, tries to close during intrauterine life but is kept open by the high pressure of the blood in it during existence in the mother.

The reason for the closing of the foramen ovale is theorized to be due to the tough membrane that overlays it in the left auricle and that this membrane acts as a "flap valve" which, under normal blood pressure, is forced over the foramen and thus closes it. It is affirmed that . . . "recent measurements of pressures within the heart affirm the above ideas."

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Influence of Shearing on Body Temperature of Lambs

A rather interesting experiment, conducted under controlled conditions on the effect of shearing of lambs on temperature and respiration, is reported from the Mississippi Agricultural Experiment Station. One group of lambs was shorn; a second group was clipped only along the back; the third group was allowed to remain in fleece. The shorn lambs had significantly lower temperatures and respiration rates than those in the other two groups. There were no significant differences in those in fleece and those shorn along the backs. The better control of body temperature is advanced as a partial explanation for more satisfactory gains in lambs shorn at the beginning of the warm season.

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Disease Spread by Raw Garbage

In a recent communication, we were requested to list swine diseases that, among other modes of infection, may be acquired following the consumption by swine of raw garbage when the latter contains pork scraps, either fresh or frozen, and pork trimmings. The following are listed for the guidance of those interested in this problem: Trichinosis, vesicular exanthema, salmonellosis, hog cholera, swine erysipelas, tuberculosis, foot-and-mouth disease, brucellosis, and anthrax.

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The cost of treatment for the relief of fringed tapeworm infestation in sheep may exceed losses due to damage caused by this parasite.—*Dean Folse, D.V.M.*

POULTRY PRACTICE PROBLEMS

W. R. HINSHAW, D.V.M., Ph.D.
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Normal Temperature of the Ostrich

In the March 1952 issue of VETERINARY MEDICINE (page 118), I discussed the susceptibility of ostriches to anthrax. Several references to the disease in ostriches were found at that time and most of them attributed susceptibility to the fact that the normal temperature of these birds more nearly approaches that of mammals than does the temperature of any other avian species. These references, however, failed to give the normal temperatures for ostriches and I could not at that time find any reference to this subject. Dr. Leonard J. Goss, veterinarian and assistant director, New York Zoological Society, has kindly furnished me with references to this information (personal letters October 30 and November 30, 1953). The references and information quoted by Doctor Goss follow:

Pembery, in Schafer's Physiology, Vol. I, page 785, gives 99.2 F. as the average of five birds. The highest of the five was 100 F.

Pickerell, of the Arizona Ostrich Farm, in a personal communication to Bergtold (Incubation Period of Birds, Kendrick-Bellomy Company, Denver, 1917) gives the following:

Female—not laying	100 F.
Female—laying	102 F.
Breeding male	104 F.

These temperatures, with the exception of that of the breeding male (104 F.), fall below those of domestic fowl, and more nearly approach the normal temperatures of mammals. The relation of the normal temperature of ostriches to their susceptibility to anthrax has not been experimentally proved.

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Washing Eggs

USDA circular 911 published in October 1952, summarizes the current information on washing and storage of eggs. A survey was made of nine different types of egg-washing machines in 31 commercial plants in seven midwestern states to determine their efficiency. Following this survey, a series of controlled experiments was conducted using four commercial egg-washing machines. A number of important conclusions were made from these studies, a few of which are as follows: (1) dirty eggs should be washed, dried, and marketed or broken promptly; (2) dirty eggs in the washed or unwashed state show greater quality decline and loss during storage than do clean eggs regardless of method of washing; (3) washed dirty eggs show greater quality decline than do unwashed dirty eggs; (4) the use of hot water is necessary and advantageous in washing of eggs; and, (5) a short presoaking or spraying of eggs with warm water containing detergent offers advantage in washing eggs.

Some important recommendations given for handling and washing of dirty eggs are: (1) handle dirty eggs promptly, (2) presoak for a short period in warm water containing detergent, (3) cleanse eggs with a washing unit that employs a scrubbing or brushing action to insure adequate removal of soil from surface of the shell, (4) sanitize the shells of washed eggs with germicidal rinse, dip or spray with hot water containing a good sanitizing agent, (5) allow cleansed eggs to dry thoroughly, and (6) candle each egg and discard all that show signs of being inedible or of doubtful value.

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Climatic stress may be a contributing factor in the development of avian monocytosis (blue comb) according to Van Ness (*Science*, 118:601, Nov. 13, 1953). His findings are based on observations in Florida where accumulated field data indicated that maximum temperatures exceeding 85 F. for three to four days increased the incidence of cases reported to the laboratory. Further data were obtained by a survey in New England in which predictions on outbreaks to occur were compared with actual cases reported to state diagnostic laboratories in the area.

Prediction accuracy in this survey indicates that there is a climatic influence on the development of the disease. The majority of the cases occurred during the hot months.

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The average size of the Pekin duck egg is 80 gm. This is slightly larger than the Muscovy duck egg which averages about 70 gm., and about 20 gm. larger than the average egg laid by a White Leghorn chicken.